B.U.S.M. Catalogued LIBRARY

The Journal of Thoracic Surgery

Official Organ
The American Association for Thoracic Surgery

Editor
EVARTS A. GRAHAM
St. Louis

Associate Editor
DUFF S. ALLEN
St. Louis

ADVISORY EDITORIAL BOARD

JOHN ALEXANDER, ANN ARBOR
EDWARD WM. ARCHIBALD, MONTREAL
LEO ELOESSER, SAN FRANCISCO
HOWARD LILIENTHAL, NEW YORK
FREDERICK T. LORD, BOSTON
RUDOLPH MATAS, NEW ORLEANS

ADVISORY EDITORIAL BOARD REPRESENTING THE SOCIETY OF THORACIC SURGEONS OF GREAT BRITAIN AND IRELAND

H. MORRISTON DAVIES, RUTHIN, NORTH WALES
J. E. H. ROBERTS, LONDON
A. TUDOR EDWARDS, LONDON

VOLUME 6
OCTOBER, DECEMBER, 1936
FEBRUARY, APRIL, JUNE, AUGUST, 1937

10692

ST. LOUIS
THE C. V. MOSBY COMPANY
1937

COPYRIGHT, 1937, BY THE C. V. MOSBY COMPANY

(All rights reserved)

Printed in U. S. A.

Press of
The C. V. Mosby Company
St. Louis

The Journal of Thoracic Surgery

VOL. 6 OCTOBER, 1936 No. 1

Original Communications

THE MUSCULAR COAT OF THE ESOPHAGUS AND ITS DEFECTS

WILLIAM LERCHE, M.D.
CABLE, WISCONSIN

A REVIEW of the structure of the muscular coat of the esophagus will be necessary, and I present a brief résumé of the work of Eduard Laimer.[1]

Laimer in describing the two layers of the muscular coat of the esophagus used the terms "the outer or longitudinal muscle layer" in contradistinction to "the inner muscle layer," pointing out that he purposely avoided using the name "circular layer" for the reason that the circular fibers are present only in a small minority.

The outer or longitudinal layer of the esophagus arises by a fibroelastic strip from the upper part of the median ridge of the posterior surface of the cricoid cartilage; it also receives muscle bundles from the inferior lateral aspect of the cricoid cartilage and a considerable amount of muscle fibers from the cricopharyngeus muscle.

These fibers do not spread out so as to surround the esophagus uniformily in its upper part, but are arranged on each side of the organ into a thick longitudinal mass. From these laterally situated longitudinal muscle masses, fibers are given off dorsally while, as a rule, in the median area of the anterior wall of the esophagus a longitudinal strip 1 cm. wide receives no cover from this source. (Fig. 1.)

The fibers first given off dorsally pass with a downward curve to unite with the fibers from the opposite side, others cross; farther down the fibers from both sides meet at an acute angle, do not cross but pass longitudinally downward. There is therefore a triangular space with point downward in the upper end of the posterior wall of the esophagus, where there are no longitudinally arranged muscle fibers. Although

Read at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

this layer of fibers often may be very thin, the inner muscle layer in the triangular space of Laimer is never without cover from the outer layer.

The distance from the inferior border of the cricoid cartilage to the lowest point of this triangle varies; as a rule, it is 2 to 3 cm., but it may be less than that or it may be 5 cm. On the other hand, it occasionally happens that the merging of the outer layer in the posterior median line first takes place at the junction of the upper and middle third of the esophagus, and, when such is the case, the median area posteriorly may have only a very thin cover of longitudinal muscle fibers.

The cricopharyngeus muscle which constitutes the lower part of the inferior constrictor muscles of the pharynx, with its lower margin, overlaps the commencement of the esophagus. Some of the fibers from this muscle cross the posterior median line to join the longitudinal layer of the esophagus on the opposite side, while others with a downward curve in a half circle embrace the esophageal wall. Sometimes the cricopharyngeus muscle extends so far down as to cover the triangular space completely.

A group of fibers from the deep layer of the cricopharyngeus muscle cross in the posterior median line of the esophagus with fibers from the opposite side, to loop around its upper end in a direction forward and downward and after crossing the midline of the anterior wall continue in a longitudinal direction.

A bundle of muscle fibers may arise separately from the cricopharyngeus muscle on the lower lateral aspect of the cricoid cartilage and run dorsally and downward to join the longitudinal muscle layer in its upper part. In four out of fifty bodies Laimer found this bundle present only on the right side; in one body it was found only on the left side. While the longitudinal muscle layer of the esophagus receives a considerable amount of fibers from the cricopharyngeus muscle; the inner (so-called circular) layer does not receive any.

Laimer quite often found the outer muscular layer in the posterior triangle too thin to give adequate support to the inner layer, and he said that under such circumstances pressure from within may cause the posterior wall to give way and bulge; and when also the cricopharyngeus muscle does not overlap the esophagus sufficiently far to give support, it makes the space still more favorable to the formation of a diverticulum.

Another area, in which the outer supporting musculature at times fails to furnish adequate cover, is described by Laimer. It is a triangular space situated more laterally between the margin of the group of muscles arising from the posterior surface of the cricoid cartilage and that of the fiber bundles of the cricopharyngeus muscle from the lower lateral aspect of the cricoid cartilage, the cartilage constituting the base of the triangle. When the cricopharyngeus muscle fails to cover this space, there is only a thin fascia covering the mucosa.

The median area of the anterior wall of the esophagus in its upper part—almost to the level of the tracheal bifurcation—is so poorly provided with longitudinal muscle fibers that in spots the inner muscle layer is not covered, and only in the minority of bodies is found a halfway strong and uniform cover of longitudinal muscle.

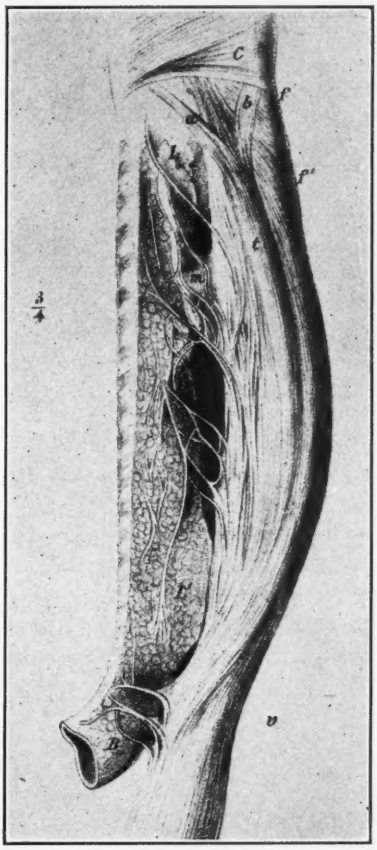


Fig. 1.—*c*, The cricopharyngeus muscle, in this case overlapping the esophagus only a very short distance. *t*, The outer or longitudinal layer. *b*, Fiber bundle from the cricopharyngeus muscle of the opposite side joining the longitudinal layer. *ff*, The fibers from the longitudinal layer joining the fibers from the opposite side to cover the inner layer in the triangular space of Laimer. *LL*, Posterior wall of the trachea with longitudinal muscle fibers to reinforce the median area on the anterior wall of the esophagus. *M*, Fibroelastic tissue connecting the anterior wall of the esophagus with the trachea. *B*, Left bronchus with bronchoesophageal muscle fibers. (Laimer.)

The fibers that arise from the ridge on the posterior surface of the cricoid cartilage and which are the main source of fibers to the outer muscular layer of the esophagus do not, as a rule, furnish fibers to cover the area in the median line of the anterior wall. When fibers from this

source are present, they are only sparsely represented. In the upper part of the anterior wall of the esophagus the fibers from the cricopharyngeus muscle, above described as crossing in the midline posteriorly

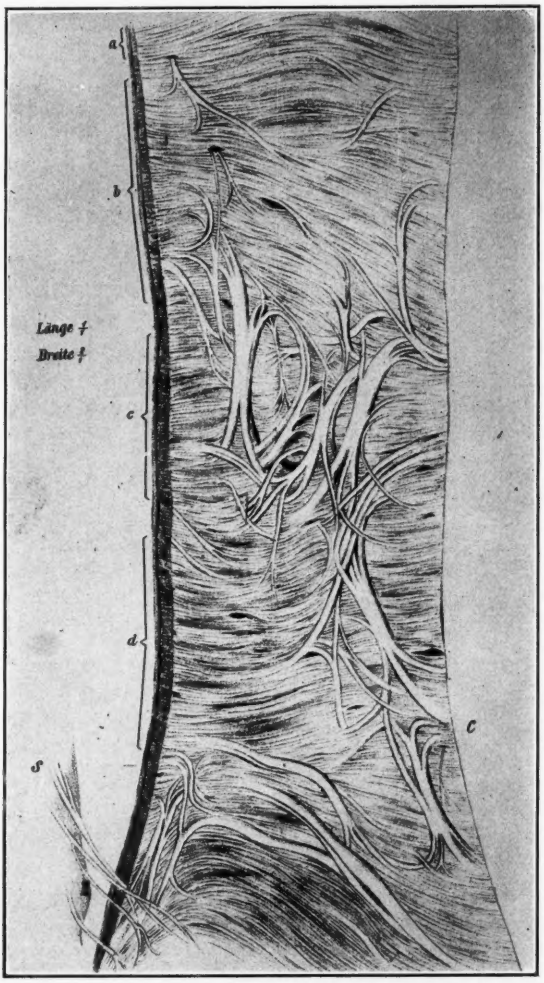


Fig. 2.—The lower section of an esophagus with unusually heavy musculature, which near the cardia was 3 mm. thick. View of the inner surface of the inner muscle layer. *a*, Elliptical fibers. *b*, Corkscrew fibers: note the distance between the levels of the upper and lower ends of the fibers. *c*, The fibers seem to be in a transitional stage to change again into *d*, screw-shaped fibers whose course is in a direction reverse to those in *b*. The fourth type of fibers is seen in bundles (like brackets). *C*, Cardia. (Laimer.)

with fibers from the other side to loop around the upper end of the esophagus and after crossing the midline anteriorly continue in a longitudinal direction, furnish part of the muscle cover for the median area.

Another source of muscle fibers for the cover of this area is the musculature of the posterior wall of the trachea. Accessory muscle fibers from the right bronchus, the aorta, and the right subclavian artery have been found. Laimer states that the esophagus may get some muscle fibers from any of the neighboring structures with which it comes in contact.

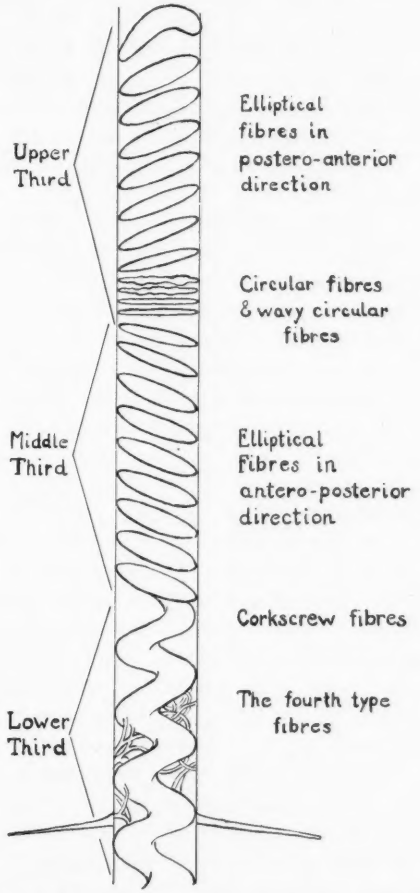


Fig. 3.—Author's diagram to illustrate the arrangement of the fibers of the inner muscle layer of the esophagus as described by Laimer.

From the foregoing it must be said that the esophagus in its upper part is surrounded by a longitudinal muscle layer with an unequal distribution of fibers, being weakest on its anterior and posterior aspects. Laterally, however, the layer is of considerable thickness, and as a rule the right side is the stoutest, as it gets more fibers from the origin on

the median ridge of the cricoid cartilage than the left and when well developed may appear triangular in cross-section.

At about the level of the tracheal bifurcation or somewhat above, these lateral muscle masses flatten out and surround the lower part of the esophagus with a quite uniform longitudinal layer, which gains in thickness as it descends, reaching its maximum strength in the lower third. Sometimes these lateral muscle masses may be seen as far down as near the cardia.

Commencing about 2 to 3 cm. above the diaphragm a membranous structure composed of fibroelastic and muscular fibers surrounds the esophagus in a tentlike manner to become attached around the margin of the esophageal opening of the diaphragm, for the fixation of the esophagus. This structure has a slit on each side for the passage of the right and left vagus nerve.

The inner (so-called circular) layer commences in the upper end of the esophagus independent of any other group of muscle fibers. It receives no fibers from the cricopharyngeus muscle.

The arrangement of the fibers of the inner layer varies in the different sections of the esophagus. In the upper third the elliptical fibers arising from the midline on the posterior wall predominate. The uppermost of these fibers from both sides of the midline first course upward, then in a curve forward and downward join each other on the anterior wall at a level below that of their starting point on the posterior wall. They run a wavy elliptical course. (Figs. 2 and 3.)

Because of the initial upward course of this first set of fibers, the inner muscular layer of the esophagus reaches its highest level on its lateral aspects. The succeeding fibers from each side of the midline on the posterior wall run in an inclined plane, surrounding the esophagus in the form of an ellipse to join each other on the anterior wall at a level 0.5 to 1 cm. below that of their starting point on the posterior wall. These fibers may occupy a section 3 cm. long or extend to the junction of the upper and middle third, their course gradually getting less oblique until they finally become circular. Some of the latter fibers run a wavy circular course. As a rule, the circular fibers are confined to a section 1 cm. long, after which they again take an elliptical course but in a direction reverse to that of the elliptical fibers in the upper third of the esophagus, namely, from the anterior wall dorsally and downward to the posterior wall. This section of elliptically arranged fibers also varies in length; it may be short or it may extend nearly to the cardia, but in the main it usually occupies the middle third of the esophagus.

In the lower third an entirely different arrangement prevails; the fibers here are winding about the esophagus in a corkscrew-shaped manner. The lower end of such a screw-shaped fiber may be on a level 0.5 to 1 cm. below that of its upper end or it may be less. These fibers mostly follow such a course to the cardia where they continue in the

innermost oblique and the circular layers of the stomach. Sometimes they end somewhat above the cardia, and a short section of the lower end of the inner layer is then composed of circular or elliptical fibers. Here and there on the lateral walls may be seen single fibers or groups of them leaving their regular course and proceeding downward in a more oblique direction than the other screw-shaped fibers.

In one instance Laimer observed in the middle third of the esophagus, on a section 2 cm. long, a layer of quite obliquely running screw-shaped fibers superimposed upon the elliptical layer on its inner surface. Small bundles of muscle fibers from the outer or longitudinal layer may join the inner layer on its lateral aspects. Near the cardia some fibers from the inner join the outer muscular layer.

When the upper half of the inner muscle layer of the esophagus is examined, there is, as a rule, no difference to be seen in the arrangement of the fibers, whether viewed from the inner or from the outer surface. In the lower half, however, there may be considerable difference between the two surfaces of the inner muscle layer; the latter on its inner surface often looks like a striped muscle plate interspersed with more or less horizontally running narrow fissures of various lengths. The striped appearance is due to the contours of the individual muscle fibers being as though they were cemented together into muscle bands several millimeters broad and joined one under another over a wide area.

Not infrequently one may see that because of the difference in the obliquity of some of the screw-shaped fibers, they may cross each other, but they all nevertheless are of the same type—namely, the screw shape. On the other hand, there may be a variety of fiber types in the same area so that screw-shaped, elliptical and more or less circular fibers are found next to each other or superimposed upon each other. On the outer surface the fibers stand out more individually and with few exceptions run quite parallel to each other.

Besides the three types of fibers mentioned above as entering into the composition of the inner muscle layer, Laimer described a fourth type which arises on the inner surface of the inner layer. These fibers are united in fine bundles and are arranged in a peculiar way entirely different from the rest of the fibers in this layer. These fiber bundles lie directly on the inner surface and penetrate the latter in every direction. They never form a continuous layer but are scattered here and there without regularity. They may run in a straight line or more or less curved or S shaped; at times they run perpendicularly in the long axis of the esophagus like brackets. Sometimes the bundles are threadlike and again they may be 1 to 2 mm. wide and more than 1 mm. thick; at times they may have sharp contours and be trabeculated. They vary in length from less than 1 cm. to 5 cm. These muscle bundles are never found in the upper third of the esophagus; they are often seen in the middle third; but they are almost always present in the lower third;

and they are most strongly represented near the narrower part of the esophagus—somewhat above the level of the diaphragmatic hiatus where by anastomosing and crisscrossing they may form a veritable network. In the narrow and muscularly strong esophagus these fiber bundles are found stouter and more numerous than in an esophagus with wider lumen and less heavy muscular coat. In an esophagus with well-developed muscular coat Laimer found its thickness near the cardia to be 3 mm. of which more than 2 mm. was due the inner muscular layer.

As the sole function of the esophagus is to convey the bolus that it receives from the pharynx into the stomach, the fibers of its muscular coat are so arranged that this performance may be carried out in the most advantageous and exact manner and at a rapid pace. The function of the outer or longitudinal muscle layer is not only to shorten the esophagus and pull it up over the bolus and to strengthen the inner (so-called circular) muscle layer, but the esophagus is suspended by the outer layer and it gives the support to the inner layer and prevents its fibers from spreading apart. It is evident that the elliptical muscle fibers are far better adapted to convey the bolus to its destination with greater speed than the circular fibers, because in contracting they shorten the tube so as to pull it over the bolus. The corkscrew fibers are still more effective, which is best demonstrated in the dog, who can bolt down a very large bolus.

A comparison between the peristaltic speed of the elliptical and circular fibers may be of interest. The speed with which the bolus moves through the cervical part of the esophagus is 20 to 25 cm. per second, in the thoracic part 5 cm. per second, and through the epicardia 1 to 5 cm. per second. In the small intestine, which has an inner "circular" muscle layer, the peristalsis moves the content at a rate of 6 to 30 cm. per hour in the fasting state, and after a meal at a rate of 70 to 120 cm. per hour or 0.00033 cm. per second.

For comparison Laimer examined the esophagus in the dog, cat, badger, rabbit, goat, calf, pig and horse. In none of these animals were circular fibers present except in the lower end of the esophagus, where often more or less circularly arranged fibers appeared. In the dog these circular fibers were massed on the cardia into a rather thick sphincter-like muscle ring. The corkscrew and the elliptical fibers predominate in these animals, particularly the former. Longitudinally running fibers appear in most of these animals only near the cardia where the corkscrew fibers take a longitudinal direction before they continue in the longitudinal layer of the stomach. In the dog, cat, and badger the entire muscular coat of the esophagus consists of two layers of corkscrew fibers winding around the organ in opposite directions and crossing each other in the median line on the anterior and posterior wall and interdigitate.

In the esophagus of the horse the corkscrew fibers likewise predominate, but in addition it has an innermost elliptical layer, and in the lower part the peculiarly arranged fibers analogous to those described as the fourth type of muscle fibers in the esophagus of man. Toward the cardia more or less circularly arranged fibers appear. Only in the lower half of the esophagus is there an outer longitudinal muscle layer. On the cardia the muscular coat has a thickness of more than 7 mm. The muscular coat of the esophagus in the rabbit has an outer longitudinal, a middle elliptical, and an inner longitudinal layer.

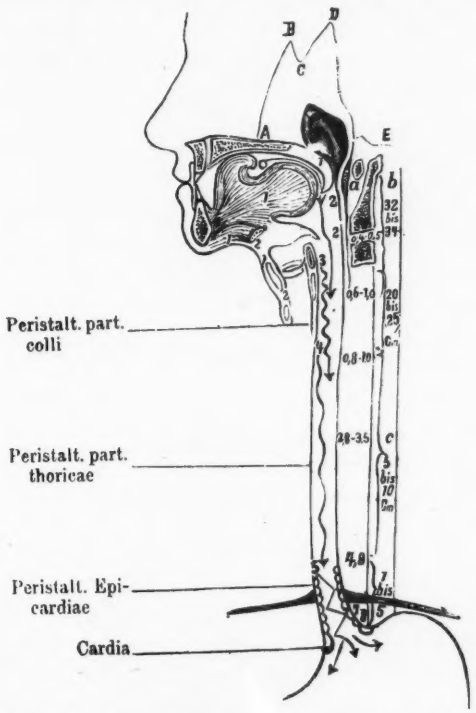


Fig. 4.—Schreiber's diagram to illustrate the mechanism of deglutition. 2, The buccopharyngeal motive power forces the bolus 4 cm. into the cervical part of the esophagus, a brief stop. 3, The swift peristalsis of the cervical esophagus carries the bolus almost to the level of the cervical bifurcation, a brief stop. 4, The slower peristalsis of the thoracic esophagus moves the bolus to the epicardia, a brief stop. 5, Peristalsis of the epicardia.

In the description of the inner muscle layer of the esophagus the peculiar arrangement of its fibers is noted. The very fact that there is such a striking difference between sections, as to the arrangement of the fibers of the inner muscle layer, suggests corresponding sectional changes in the functional activities; hence a brief review of the modus operandi of the mechanism of deglutition is of interest. According to

Schreiber[2] the bolus resting on the back of the tongue is forced behind the anterior pillars of the fauces chiefly by the mylohyoid muscle, while simultaneously complete closure toward the mouth and nasopharynx is effected; with the added force of the hyoglossus and the thyrohyoid muscles the bolus is pressed deeper into the pharynx, from where, after the opening of the upper end of the esophagus has been completed by the contraction of the geniohyoid and the relaxation of the inferior constrictors, the middle constrictor muscles force the bolus into the cervical part of the esophagus—for a distance of 4 cm. (Fig. 4.)

Here the buccopharyngeal force is exhausted; the bolus comes to a stop for a fraction of a second, and a new motive energy—the swift peristalsis of the cervical esophagus, which begins while the force of the middle constrictors is moving the bolus into the cervical part—now carries it into the thoracic region as far as the lower part of the upper third of the esophagus, almost to the level of the tracheal bifurcation. At this station not only is there a momentary stop but there is a change in the velocity of the bolus, pointing to a shift in the moving force, namely, to the slower and more irregular peristalsis of the thoracic esophagus which now carries the bolus to the epicardia where it rests for 0.3-0.5 second before the peristalsis of the epicardia moves it into the stomach.

There seems to be a definite relationship between the rate of the peristaltic speed and its stops as found by Schreiber and the sectional arrangement of the muscle fibers of the inner layer as described by Laimer.

In this connection mention should also be made of the constrictions which are found in more than 50 per cent of gullets. The three constrictions most constantly present are at the beginning of the organ or somewhat below, at the level of the tracheal bifurcation or somewhat above, and at or a little above the level of the diaphragm. The muscle fibers in the upper third of the esophagus are mostly of the striated variety, in the lower two-thirds nonstriated.

CASE REPORTS

CASE 1.—A male, aged fifty-one years, gave the history of injury to his back with fracture of three ribs at the age of forty-six years; he had had typhoid fever three years later. Said he gets hives when his digestion is upset. Constipation. A highly nervous individual.

His complaint was difficulty in swallowing of four months' duration. He first noticed that solid food would stick in his throat. Somewhat later he found that of two pills he had taken, one came up two hours later. His dysphagia gradually increased, and on the morning of consultation he found it difficult to swallow liquid.

Esophagoscopic examination revealed a small diverticulum in the upper end of the posterior wall of the esophagus, in the posterior triangle of Laimer.

X-ray plate, Fig. 5, shows the diverticulum.

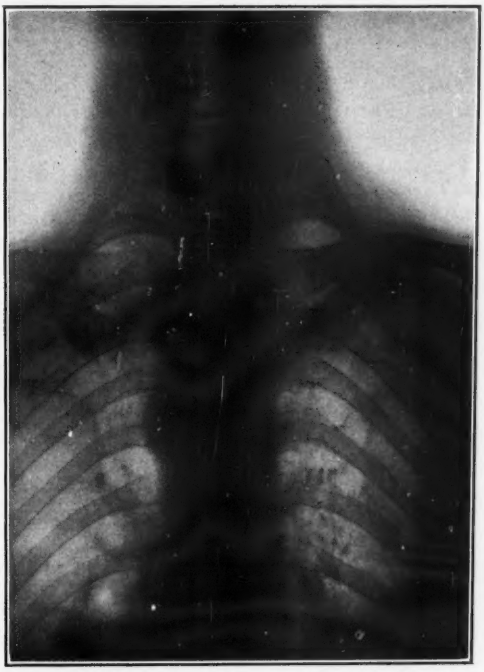


Fig. 5.—Case 1, diverticulum in the posterior triangle of Laimer.

Fig. 6.—Case 2, diverticulum in the lateral triangle of Laimer.

CASE 2.—Male, aged sixty-three years, had had thyroid enlargement for many years, with a retrosternal extention.

His chief complaint was difficulty in swallowing of several years' duration. He would often choke on a bit of dry food or a small piece of apple. He had to drink slowly.

X-ray examination showed a diverticulum to the right of the midline, also thyroid enlargement. (Fig. 6.)

Esophagoscopy: The opening into the diverticulum was seen on the right side —in the lateral triangle of Laimer.

A diverticulum in both the lateral and the posterior triangles of Laimer may be present in the same patient.

CASE 3.—A male, aged fifty-five years. About four years previous to consultation the patient first noticed an occasional choking sensation on eating. Two years later, after eating dried beef, he had a spell when he could not swallow anything for three days. Since then he has had difficulty on eating solid food unless he takes plenty of liquid at the same time.

Fig. 7.—Case 3, large diverticulum of the anterior wall of the esophagus with a series of small diverticula somewhat above.

The x-ray picture in Fig. 7 shows a large diverticulum of the anterior wall of the esophagus. The shape of the notches some distance above this diverticulum indicates the direction of the elliptical muscle fibers. Between these fibers are seen small accumulations of barium mixture held in the minute protrusions of the esophageal mucosa—which are pressure diverticula in the making.

Esophagoscopy: There was no opening into the diverticulum to be seen. After considerable search a slight depression in the mucosa, rectangular in outline and in the long axis of the esophagus, was seen on the anterior wall just above the level of the tracheal bifurcation. On this depressed area, which was about 3 cm. long and somewhat less than 1 cm. wide, longitudinal mucosal folds stood

out against the surrounding smooth mucosa and caught my attention. I mention this because it might easily have been overlooked. I have examined quite a number of diverticula through the esophagoscope; I have seen a small pressure diverticulum in the lower third of the esophagus, in which the mucosal pouch would in part pout into the lumen of the esophagus, but I have never seen prac-

Fig. 8.

Fig. 9.

Fig. 8.—Case 4, showing the corkscrew fiber bands of the esophagus.
Fig. 9.—Case 4, in a state less firmly contracted the fiber bands have the appearance of an auger.

tically every trace of a large diverticulum vanish as soon as it had emptied itself of its contents. It may truly be said to be a phantom diverticulum, and shows the remarkable elastic resiliency of the esophageal mucosa and submucosa.

This diverticulum was in the median area on the anterior wall of the esophagus where, according to Laimer, only in the minority of bodies is found a halfway strong and uniform cover of longitudinal muscle fibers.

CASE 4.—A male, aged fifty-three years, had some difficulty swallowing off and on for four years. At times he had a sensation of fullness in the throat and difficulty in swallowing water; he had to hold liquids in his mouth for a while before he could swallow them. Finally he had trouble in swallowing both liquids and solid foods.

Fluoroscopic examination: Thin barium mixture passed rapidly through the esophagus. In the lower part of the organ the corkscrew muscle bands came into view, more prominently so after the second or third swallow (Fig. 8). When thick barium mixture was given, the fiber bands had an auger-shaped appearance (Fig. 9).

Laimer described the appearance of the inner surface of the inner muscle layer of the esophagus in its lower half as follows (Fig. 2): "It looks like a striped muscle plate interspersed with more or less horizontally running narrow fissures of various lengths. The striped appearance is due to the contours of the individual muscle fibers being as though they were cemented together into bands several milli-

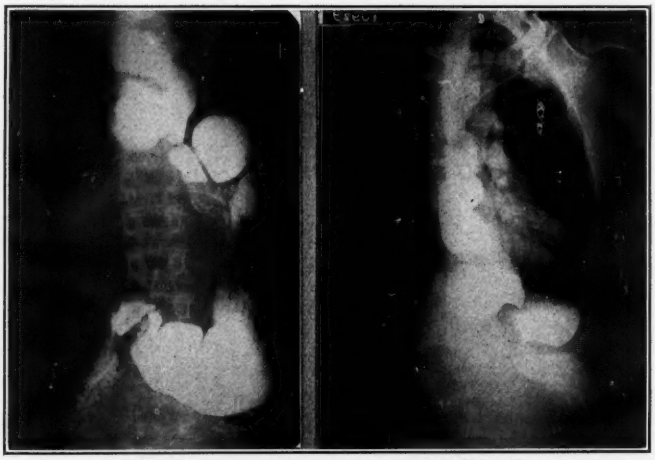


Fig. 10. Fig. 11.

Fig. 10.—Case 5, x-ray photograph of esophagus with multiple diverticula in anteroposterior view.

Fig. 11.—Case 5, x-ray photograph of esophagus with multiple diverticula in lateral view.

meters broad and joined one under another over a wide area." The muscle bands so well described by Laimer are shown in Figs. 8 and 9.

The fissures referred to (shown in Fig. 2) and found only in the lower half of the esophagus are not further mentioned in the work of Laimer. Do these fissures have any significance in relation to the pressure diverticula occurring in the lower part of the esophagus?

CASE 5.—A male, aged thirty-eight years, had been in good health until the age of ten years, when after eating frozen pears he was taken violently ill. He had severe cramps in the abdomen and was forced to roll about on the ground in great agony before he was able to go home. Following that he was sick in bed for two weeks, and since then he had always had difficulty in swallowing and eating solid food and he found it necessary to take a large amount of water to wash it down.

X-ray photographs (Figs. 10 and 11) show multiple diverticula of the esophagus. Six years later, at the age of forty-three years, the patient returned complaining of loss of weight, loss of appetite, and progressive fatigue.

Diagnosis: Malignant growth of one of the diverticula with involvement of the liver.

The autopsy (Fig. 12) was made by Dr. Margaret Warwick from whose report I quote: "The esophagus is very much dilated, presents a very markedly thickened wall and contains three diverticula, all situated just above the

Fig. 12.—Case 5, photograph of specimen of esophagus with multiple diverticula.

diaphragm. The largest of these is near the diaphragm, is thin walled and with no tumor tissue. The smallest diverticulum is directly across from the largest one, also just above the diaphragm—has tumor masses around it. The third diverticulum is situated somewhat higher, has tumor tissue in its floor typical of carcinoma. Between the two lower diverticula there is only a very small narrow lumen which leads into the stomach. It seems impossible that any food could escape the diverticula on either side and pass through this small passageway into the stomach."

The specimen of this esophagus was recently examined microscopically by Dr. E. T. Bell, who found that the largest diverticulum has a wall composed only of mucosa and submucosa; the other two diverticula have all the layers of the wall of the esophagus on the sides and only carcinoma in the base. The two lower diverticula are situated opposite each other at the very inlet to the hiatus oesophageus, and as expressed by Dr. Warwick in the autopsy report "it seems impossible that any food could escape the diverticula on either side and pass through this small passageway into the stomach." No matter how small the two lower diverticula might have been, placed as they are, they would have given rise to symptoms. In view of this and the fact that the patient had been in perfect health up to the age of ten years, when the trouble with his gullet began suddenly after the ingestion of the frozen pears, the possibility that these diverticula might be congenital in origin can positively be excluded.

The diverticula in this case were caused by a sudden strong continuous spastic contraction of a section of the esophagus in its lower third including the epicardia; the spastic contraction was precipitated by the ingestion of frozen pears—perhaps not merely acting as a foreign body but probably by the cumulative action of the thermal, the chemical, and the mechanical stimuli provided by this agent. Two of the diverticula have all the layers of the wall of the esophagus on the sides and only carcinoma in the base. The largest diverticulum has a wall composed only of mucosa and submucosa. The initial bulge of the three diverticula probably started at the same time, but the fibers of the muscular coat in the largest one spread apart and permitted the mucosa and submucosa to protrude, thus providing the safety valve, and relief of the intraesophageal pressure.

These three diverticula were caused by abnormal intraesophageal pressure in the lower part of the esophagus, where the muscular coat is heaviest and where no congenital defects have been described except the fissures referred to above and shown in Fig. 2. It is logical to assume that the great intraesophageal pressure in this case would be most effectively exerted on the areas of least resistance —the fissures.

These fissures may also partly explain the occurrence of the more or less symptomless diverticula in the lower third of the esophagus.

This case occurred in the practice of Dr. E. T. F. Richards, St. Paul, Minn. I was consulted about the x-ray findings at the time. Dr. Richards kindly turned the material over to me for use in this paper.

The interest in the areas of the muscular coat of the esophagus where deficiency in fibers mostly occurs, has been chiefly focused on the triangular space on the posterior wall described by Laimer in 1883, who also stated that he had often (not always) found this space only thinly covered by fibers from the longitudinal muscle layer, and that, when such is the case, this space would be favorable to the formation of a diverticulum. In 1891 Haeckermann[3] described a museum specimen of a diverticulum, in which the musculature showed that it had its origin in the posterior wall of the esophagus, in the triangle of Laimer, which he refers to as the "anlage."*

One of the functions of the outer or longitudinal muscle layer of the esophagus is to support the inner muscle layer and to prevent its fibers from spreading apart. When it fails in this, the fibers of both layers

*In a number of articles on diverticula of the esophagus, the posterior triangle of Laimer has been referred to as the "Lannier—Hackerman" triangle, which is simply a misspelling of Laimer's name, as well as that of Haeckermann's.

may give way and permit the protrusion of the esophageal mucosa and submucosa between the fibers. Intraesophageal pressure is the cause of such a mucosal protrusion, hence the name pressure diverticulum.

Certain areas have been described in this paper in which a deficiency in muscular cover may occur:

1. The posterior triangle of Laimer.
2. The lateral triangle of Laimer.
3. The median area on the anterior wall.
4. The median area on the posterior wall.
5. The fissures in the lower half of the esophagus.
6. The esophagus with large lumen and a thin muscular coat.

Although the deficiency in muscular cover in these areas is congenital, the mucosal protrusions occurring there are probably never congenital.

Pressure diverticula may occur in any part of the esophagus, but there are three sections that seem to be seats of predilection: (a) the section corresponding to the posterior triangle of Laimer; (b) the median area on the anterior wall above the level of the tracheal bifurcation; and (c) the lower third of the esophagus above the diaphragm. In these three sections congenital defects in the muscular coat occur; in these three sections the muscle fibers of the inner layer are arranged in a manner peculiar to the section and different from that of the preceding section; and in these three sections are the stations where the bolus normally comes to a short stop and where a change in the motive power takes place. May this change in functional activity render these sections more susceptible to irritation?

A local muscle spasm is probably the agent giving rise to the pressure that causes the mucosal protrusions. In a paper[4] published in 1915, I made the statement that "a local neurosis is present in every case of anatomical lesion of the gullet—at some time"—that is, a local neuromuscular disturbance, one expression of which is a local muscle spasm. A bolus of food, the temporary lodging of a foreign body in the gullet, an obstruction in the tube itself or encroachment of a neighboring structure, etc., may likewise incite local muscle spasm in the esophagus, as may a reflex from an abdominal organ. On the other hand, there is a difference in the physiologic action of the two muscle layers of the esophagus, and it seems possible that local neuromuscular imbalance between the two may occur, so that while the inner layer in a segment is holding the bolus in spasm, the outer layer, one of whose functions it is to prevent the spreading apart of the fibers of the inner layer, may be in a state of relaxation and permit bulging. Carlson and Luckhardt[5] in certain experiments on turtles found that hypertonus of the esophagus mainly involving the inner (or circular) muscle layer may take place without shortening of the esophagus, and that the hypertonus

thus induced is rarely equal in degree throughout the entire length of the organ—it permits also development of local spasms of the esophagus. If this also applies to man, my theory may be tenable.

The mucous membrane of the esophagus, according to Boring,[6] is sensitive to thermal, mechanical, chemical (to alcohol and hydrochloric acid), and electrical stimuli, and more so in the upper part than in the lower. A bolus of food may for various reasons be retarded in its passage through the esophagus and act as an irritant; it may be too large, too small, too dry, or too hot. A swallow of a cold carbonated beverage or ice cold water drunk too fast may give a feeling of contraction or a sense of choking, which soon passes off. Such a sensation may be due to a slight local muscle spasm in the esophagus, or spasm plus relaxation, which may initiate the first slight bulging of the wall, especially in a person predisposed by a congenital defect in the muscular coat, and gradually develop into a diverticulum.

It seems therefore that there are two ways in which diverticula may be produced by intraesophageal pressure:

1. Acute—where the stimulating agent precipitates a sudden strong and continuous spastic contraction in a section of the esophagus, whereby the fibers of both muscle layers are forced apart permitting the protrusion of the mucosa and submucosa; or by forcing the entire esophageal wall in a circumscribed area.

2. By a more gradual process—by repeated slight transitory muscle spasms of a segment of the inner muscle layer, while the outer muscle layer remains in a state of relaxation. Once started, a slight bulging in the esophageal wall becomes an irritant, and thus a vicious circle is established.

In comparing the incidence of diverticula of the esophagus in man with that of the horse, we find that out of twenty-six cases of diverticula of the esophagus in the horse, according to Rubeli,[7] eleven cases were found above the cardia close to the diaphragm; seven cases were in the thoracic part and eight cases in the cervical part. Most of the latter were found in the lower cervical section. The lumen of the esophagus in the horse is narrowest at the cardia and in the lower cervical section. The musculature is heaviest at the cardia, averaging 12 mm. in thickness, and for a distance of 7 cm. above the cardia it is 10 mm. in thickness. In the lower cervical section, where the majority of the diverticula in that region occurred, the musculature is thicker than in the adjoining sections above and below. It seems that in the horse as in man there are seats of predilection for diverticula of the esophagus, but why are these seats located where the musculature is the heaviest and the lumen the narrowest? The answer may be the same as that I propounded in the case of man, namely, a change in the functional activity in these sections, rendering them more susceptible to irritation with consequent precipitation of local muscle spasm or spasm plus relaxation.

SUMMARY

1. Congenital defects which have been found to occur in the muscular coat of the esophagus are described.

2. Sectional differences in the arrangement of the fibers of the inner muscle layer and

3. Corresponding changes in the functional activities are discussed.

4. Pressure diverticula may probably occur in any section of the esophagus irrespective of the thickness of the muscular coat, depending upon the stimulus.

5. A congenital defect in the muscular coat is an important predisposing cause in pressure diverticula of the esophagus.

6. The three segments in which the conditions referred to in 1, 2 and 3 are found are also the principal seats of pressure diverticula of the esophagus.

7. Functional irritation inducing local muscle spasm, we believe, is more easily incited in these three segments for reasons stated.

8. The local muscle spasm may effect a pressure diverticulum (a) by a sudden, strong and continuous spastic contraction of a segment of the esophagus, or (b) by a slow process of repeated slight and transitory spasms of a segment of the inner muscle layer, while the outer muscle layer remains in a state of relaxation.

REFERENCES

1. Laimer, Eduard: Beitrag zur Anatomie des Oesophagus, Medizinische Jahrbücher, Wien. Jahrg., pp. 333-388, 1883.
2. Schreiber, Julius: Ueber den Schluckmechanismus, Berlin, 1904, August Hirschwald.
3. Haeckermann, Karl: Beitrag zur Lehre von der Entstehung der Divertikel des Oesophagus, Dissertation, Göttingen, 1891.
4. Lerche, William: Diseases of the Esophagus, Studied by the Aid of the Esophagoscope, New York M. J. **101**: 1266, 1915.
5. Carlson, A. J., and Luckhardt, A. B.: Studies on the Visceral Sensory Nervous System. X. The Vagus Control of the Esophagus, Am. J. Physiol. **57**: 299, 1921.
6. Boring, E. G.: The Sensations of the Alimentary Canal, Am. J. Psychol. **26**: 1, 1915.
7. Rubeli, O.: Ueber den Oesophagus des Menschen und verschiedenen Hausthiere, Berne, 8°, 1889.

DIAGNOSIS AND TREATMENT OF BENIGN ULCERS OF THE ESOPHAGUS, WITH CASE REPORT

H. Ryerson Decker, M.D., F.A.C.S.
Pittsburgh, Pa.

BENIGN ulcers of the esophagus may be said to include all ulcers that are nonmalignant. Some, however, are kindly only in name, since they are responsible for persistent disturbance of function and alteration of structure that lead to disability and invalidism, and at other times to death by reason of hemorrhage and perforation. For this reason alone they merit a more serious consideration by the clinician that perhaps has been accorded in the past.

A case of ulcerative esophagitis is reported, in which recovery has not occurred after four years of treatment.

W. B., male, aged forty-three years, was admitted to the Presbyterian Hospital May 17, 1932, complaining of epigastric pain. This had been present in variable degrees for fourteen years. The onset came while he was in army service in France in 1918. He was hospitalized there three separate times on account of this pain, with a diagnosis first of gastroenteritis and later, abdominal influenza. In 1919 on discharge from the army his family physician treated him for a time for duodenal ulcer with amelioration of symptoms, but not complete relief. Until 1930 the pain was intermittent, but in the last two years it has been of nearly daily occurrence. Now it has become sharp and severe, is located in the epigastrium, and awakens him from a sound sleep. It is relieved by food, especially milk and soda. If more than the usual amount of food is taken at one time, there is a bloated feeling in the epigastrium. He has been constipated. There has been no vomiting, nor hematemesis nor melena. He has lost about 20 pounds in weight, and considerable strength.

Past History.—Measles, mumps, pertussis, and typhoid fever in childhood. He smokes about ten cigarettes a day. He is married, and has one child aged fourteen years. Father died of Bright's disease. Mother living and well.

Examination.—Height 5 feet, 9 inches; on admission to the hospital, weight 130 pounds. He was somewhat undernourished, but did not look seriously ill. Teeth—several were carious and several had apical infection. Heart and lungs normal. Temperature was normal. Pulse 72; respirations 18. Blood pressure—systolic 122, diastolic 76. Abdomen—soft and flat. There was tenderness to deep pressure at the umbilicus, and above, and to its outer side. Tendon reflexes were normal.

Laboratory Findings.—Urinalysis normal. Hgb. 90 per cent; R.B.C. 4,600,000; W.B.C. 10,300; polymorphonuclears 67 per cent; small mononuclears 32 per cent; mast cell 1 per cent. Wassermann test negative. Kahn test negative. Gastric analysis—Ewald test meal—free acid 36; combined 12; total 48. Blood absent. Einhorn string test negative.

X-ray Examination.—(Dr. G. W. Grier.) Stomach is rather large but normal in outline. There is very extensive spasm at the pylorus, but the stomach empties in six hours. Duodenum is never filled. On account of constant deformity of the

Read at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery, Rochester, Minn., May 4-6, 1936.

duodenum there may be a duodenal ulcer. There is no evidence of a pathologic condition in the small intestine or the colon. The appendix is not filled.

Diagnosis was made of duodenal ulcer, and surgery was decided upon. Operation May 20, 1932. Spinal anesthesia. Excision of the ulcer. Pyloroplasty after the method of Horsley, and appendectomy. Findings—a large ulcer at the pylorus involving the upper two-thirds of the anterior wall, a small diverticulum of the anterosuperior duodenum just beyond the ulcer. Appendix looked chronically diseased. Pathologic reports were simple ulcer and acute appendicitis.

Postoperative Course.—On the evening of the second day there was emesis of 200 c.c. of brownish fluid, and again during the third night. The patient complained of epigastric pain and distended feeling in the upper abdomen, so a gastric lavage was instituted daily. At the time of each lavage a quantity of foul-smelling brown fluid was evacuated. Temperature was normal, but pulse became somewhat elevated.

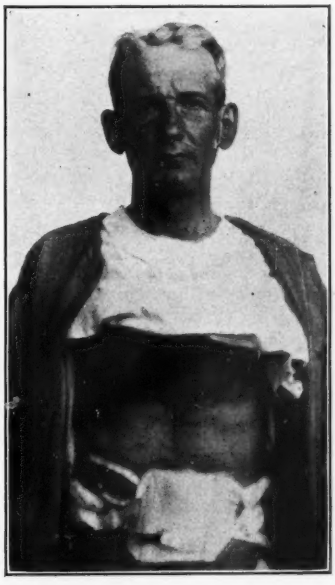


Fig. 1.—Case of benign ulcer of esophagus secondary to acute esophagitis.

Evidence of gastric distention persisted, suggesting that there was definite obstruction at the pylorus, without any evidence of peritonitis. On May twenty-seventh, on week after the first pyloroplasty, the abdomen was reopened. The omentum was found adherent to the abdominal incision and also to the pyloroplasty. The stomach was distended, and there did not seem to be free patency of the pylorus. A posterior gastroenterostomy was performed. The postoperative course was now smooth. There was no vomiting or distention. In ten days the patient was taking a selected soft diet. On June 9 he was sent to the dental department where his diseased teeth were extracted under novocaine anesthesia. On June 10, for the first time, he complained of difficulty in swallowing toast and mashed potatoes without the aid of fluid. There was no pain during or after the swallowing. He was discharged from the hospital two days later with the abdominal incision healed. He said he felt exceptionally well. The dysphagia was still present in a slight degree, but he made so light of it that an esophageal investigation was not thought necessary.

Subsequent Course.—After leaving the hospital the dysphagia became worse. He had to wash solid foods down. On June 15 a piece of toast soaked in milk stuck, seemingly in his throat, and was expelled. From then on he took nothing but liquids which passed readily until June 20 when eggnog and water alone could not be swallowed, and were regurgitated. Obstruction seemed to be released in several hours, and he could swallow water. At this juncture he reported back to the hospital, and was readmitted.

X-ray Examination.—(By Dr. Grier.) Barium capsule stops in the esophagus about two inches above the cardiac opening. There is very violent obstruction in the esophagus at this point.

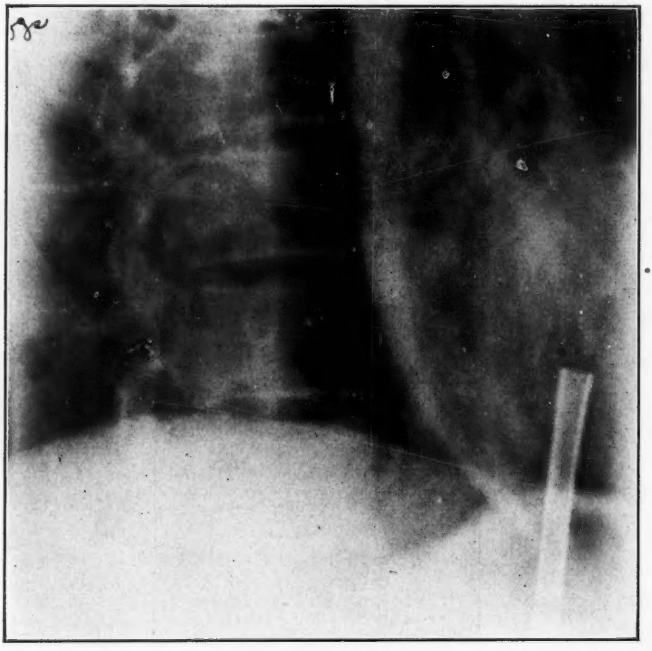


Fig. 2.—X-ray of esophagus after four years of treatment. Showing narrowing of terminal esophagus. Gastrostomy tube is in place.

Esophagoscopy.—(By Dr. Ellen J. Patterson.) June 21, at 35 cm. to 40 cm. from the incisor teeth beginning at the level of the diaphragm was a cauliflower growth several centimeters in extent, involving the whole circumference of the esophagus, which bleeds easily and has the appearance of malignancy. A specimen was removed for examination, which on micropsy by Dr. Wallhauser proved to be necrotic granulation tissue with no evidence of malignancy. On suggestion of Dr. Patterson, a Stamm-Kader gastrostomy was performed under novocaine anesthesia on June 28. There were adhesions of the omentum to the anterior parietes, and in one place to the stomach itself near the greater curvature. These were freed, but no further exploration was carried out. On July 5, an 8 mm. esophagoscope was introduced to the cardia. There were inflammation and ulceration above the cardia, but much less marked than previously. Bougies were passed through to the stomach. Chlorlyptus oil was applied to the ulcerated area. On July 8 the treatment was repeated. On

July 12, esophageal mucosa was still inflamed above the cardia. Ulceration and granulations bled very easily. A No. 22 bougie was passed through the cardia. Patient was discharged from the hospital on July 18, 1932, to report back for esophageal treatments by Dr. Patterson. At this time he was taking fluid both by mouth and through the gastrostomy tube. He has reported back for treatments faithfully for a period of four years, at first once a week, and now once a month. The ulcer itself was healed in about three months, leaving some narrowing of the lower end of the esophagus without evident scarring. The narrowest portion according to recent x-ray examination is about 1.5 inches above the cardiac orifice. There is no evidence of displacement or twisting of the esophagus, nor of dilatation above. Barium meal goes through without difficulty as does an 8 mm. esophagoscope. From the first there has been spasm at the cardiac orifice definitely seen and felt during the passage of bougies into the stomach. It has varied in degree from one treatment to another, and occasionally has been practically negligible. His dysphagia has continued without improvement, from his own standpoint, in the last three years. There has been no difficulty at all with liquids, but solid foods must be ground fine and washed down. At times, even so, there may be complete blocking, which will open in an hour or two if he rests quietly and relaxes. There has been no pain at any time. He has been working on a small farm, and his general health has been excellent.

Examination.—April 24, 1936. Weight 142 pounds. Urinalysis normal. Hgb. 94 per cent; R.B.C. 4,800,000; W.B.C. 9,300; polymorphonuclears 69 per cent; small mononuclears 25 per cent. Fractional gastric analysis (after Ewald test meal —specimens obtained every fifteen minutes through a gastrostomy tube).

Free	5	Total 35
30 minutes	56	68
	62	76
60 minutes	50	62
	36	48
90 minutes	42	50

Microscopic examination—no blood.

X-ray examination of the stomach shows very rapid emptying through the gastroenterostomy stoma. No part of the meal goes through the pylorus. Duodenum is not seen. X-ray examination of the thorax shows no abnormality in the heart or lungs. There is one adhesion evident between the pleura and the left diaphragm. Esophagoscopy—April 24, 1936, Dr. Patterson introduced a No. 30 bougie retrograde through the gastrostomy opening without difficulty. The gastrostomy has never given any trouble, except at times skin irritation. It has been maintained as a precautionary measure, but it has not been used for feeding for over two years.

Summary.—A case of ulceration of the esophagus is presented, which developed subsequent to a pyloric ulcer of long standing. It was thought at first glance in view of this association that it might be a peptic ulcer, but its gross appearance, confirmed by microscopy, was that of an ulcer secondary to esophagitis. This in turn may have been induced by vomiting and by the passage of a stomach tube. While the ulcer healed fairly promptly without mucosal scarring, there is now a narrowing of the lower end of the esophagus at the diaphragmatic level, which can be accounted for by a fibrosis in the esophageal wall. There is also a persistent spasm at the cardiac orifice. It is apparently this spasm that is responsible for the continued dysphagia. Dr. Patterson has felt that there is a definite psychologic factor to account for the spasm, though the man seems outwardly and by any neurologic test to be nervously stable. I am more inclined to think the spasm is a reflex from the stomach, due to the presence of adhesions secondary to the surgical operations, or of the gastrostomy tube, or of a marked hyperacidity which is present, and incidentally

is higher than prior to the gastroenterostomy. At one time in his treatment belladonna and bromides were given him to a point of tolerance without any influence on the spasm. Bouginage has given only partial relief. I believe the next indications are to remove the gastrostomy tube and to treat the gastric hyperacidity. This case illustrates how benign ulceration of the esophagus may lead to troublesome sequelae that baffle successful treatment.

Judging by the comparatively meager reports in the literature and our own observation, benign ulceration of the esophagus occurs rather infrequently. Chevalier Jackson[1] in 4,000 endoscopic examinations of the esophagus found 21 peptic ulcers and scars of 67 others (2.2 per cent). At the Tuberculosis League Hospital in Pittsburgh no cases of tuberculous ulceration of the esophagus have been recognized clinically, nor at necropsy in 6,176 admissions for tuberculosis. At the Presbyterian Hospital in 48,243 consecutive admissions benign ulceration was diagnosed six times. In a series of 10,000 autopsies at the University of Leeds reported by Stewart,[2] only one peptic ulcer was found. Bartels[3] reported that in 6,000 necropsy specimens of the esophagus at the Mayo Clinic acute ulcerative esophagitis was found 82 times (1.3 per cent). Lockard[4] reported 25 tuberculous esophageal ulcers in 16,489 autopsies. In a group of four Pittsburgh hospitals benign ulceration of the esophagus was found 18 times in 4,382 necropsies (0.4 per cent). Pathologic diagnosis in this group was made as follows: acute esophagitis 6; varicose veins 4; mercurial poisoning 2; aneurysm of aorta 3; syphilis 1; diphtheria 1; diverticulitis 1.

Classification of ulcers of the esophagus based on etiology.

A. *Benign.*

1. Traumatic.
   a. Foreign bodies.
   b. Instrumentation.
   c. Decubital.
   d. Burns (corrosives).
2. Acute inflammations.
   a. Esophagitis.
   b. Diverticulitis.
   c. Infectious disease.
      Scarlet fever.
      Typhoid fever.
      Pneumonia.
      Diphtheria.
3. Chronic inflammation.
   a. Tuberculosis.
   b. Syphilis.
   c. Blastomycosis.
   d. Actinomycosis.

4. Peptic.
5. Varicose.
6. Papilloma.

B. *Malignant.*

Pathogenesis.—The pathogenesis of ulceration of the esophagus requires little comment. No one factor is solely responsible. Anatomically the gullet is in a vulnerable position between the oral cavity and the pharynx above, furnishing a constant bath of bacteria, and the stomach below regurgitating gastric juice on little provocation. Acute esophagitis with ulceration is prone to develop in debilitated individuals, or those who vomit considerably, e.g., alcoholic individuals. The influence of gastric juice is unquestionable, not only in the formation of these ulcers but also in their perpetuation. Trauma from the passage of instruments or from a stomach tube is not a prolific factor, nor are lymphatic and hematogenous infections, spreading from intra-abdominal disease and lesions elsewhere in the body. These, however, may reflexly produce spasm of the esophageal wall. This in turn leads to food retention and stagnation, which predisposes to local maceration of the mucosa and ulceration.

Some interest attaches to the pathogenesis of peptic ulcers. Inasmuch as they are found at times in association with gastric and duodenal peptic ulcers, one might consider the same factors to be concerned in their development. Regurgitation of acid content of the stomach and the presence of heterotopic gastric mucosa (if it is really not normal gland bearing esophageal mucosa) are undoubted factors. Jackson[1] has made out a strong case for infection of the mouth, nasal sinuses, and throat as the active factor. Thus, he found that in 21 patients having acute peptic ulcer, 19 (80 per cent) had a chronic infection in the mouth, tonsils, and sinuses. In 67 cases of healed peptic ulcer 59 patients (88 per cent) had diseased tonsils, or had had a tonsillectomy; 3 had a foul mouth with carious teeth. In a control series of 88 foreign body cases only 14 (15 per cent) showed similar infection. Jackson believes that neither cardiospasm nor insufficiency of the cardia, nor other intraabdominal diseases are factors. In experimental work with dogs, Friedenwald[5] found that mucosal and penetrating ulcers healed very rapidly. Applications of 10 per cent hydrochloric acid to an acute ulcer might produce a chronic ulcer, but normal esophageal mucosa was not affected by the acid. One cannot overlook the neurogenic factor in the production of peptic ulcers in view of Cushing's painstaking and convincing presentation of the rôle midbrain plays in the pathogenesis.

Pathology.—Ulcers may develop in any part of the esophagus, either solitary or multiple. There is predilection for the formation in the lower one-third, and particularly just above the cardiac orifice of the

stomach in the diaphragmatic area. Most heal quickly and leave little or no scarring. Some become chronic, and heal with scarring, with or without stricture. A few, not particularly of any one type, perforate. Then there is set up an infection of the mediastinum or respiratory tract. Fistulous tracts may form into the pleural spaces, trachea, lung, bronchi, pericardium, or neck. Occasionally there is perforation into one of the large thoracic blood vessels, particularly the aorta. Perforation on the other hand may be due to extrinsic disease, such as suppurating lymph nodes or aneurysm. A question of perhaps more academic than clinical interest is concerned with the formation of a malignant ulcer from a benign one. Ortman in 1901 and Tileston[6] in 1906 reported the development of carcinoma in a scar from an old peptic ulcer. Guisez[7] reported a case of epithelioma at the site of a lye stricture.

Symptomatology.—Subjective symptoms are frequently absent. When present they are not distinctive of any one type of ulceration, nor are they to be distinguished from those of esophagitis without ulceration. Dysphagia and pain are early in appearance. Pain is cervical or substernal or epigastric, depending upon the level of the ulcer, and has a definite tendency to radiate into the back. At times there is only odynophagia. There may be heartburn, regurgitation or vomiting. There is bleeding in at least 50 per cent of ulcer cases, recognized usually as melena, for hematemesis is rare. Hemorrhage may be profuse and lead to fatal issue. If perforation of an ulcer takes place, there may be severe pain and shock. The clinical picture in low ulcer simulates perforation of a gastric ulcer. Severity of the symptoms is likely to depend upon whether the perforation takes place suddenly or gradually. The subsequent picture depends upon the viscus or space into which the perforation has taken place. Thus, if into the pleural cavity, a suppurative pleurisy or pneumothorax develops; if into the mediastinum, a mediastinitis; if into the neck, cellulitis; if into the lung, pneumonitis. These two factors, hemorrhage and perforation, are the liabilities in benign ulcers that are life ending.

Diagnosis.—Diagnosis of ulceration of the esophagus will be suggested by the subjective symptoms. It will be substantiated in a number of cases by x-ray examination. It will be established with certainty in most cases by endoscopy.

X-ray examination will not demonstrate many benign ulcers, only those that are deep enough to cause deformity or stricture. When there is delay in the progress of the barium meal, due to stenosis or spasm, ulceration may be only suspected.

Esophagoscopy has become indispensable to diagnosing esophageal lesions. Biopsy in addition is needed, since the gross appearance of the ulcers as seen through the esophagoscope may not be distinctive.

The examination should be made by a well-trained endoscopist. In the best of hands at times ulcers are hard to find, especially if bleeding has been started by the instrument. Nor is the procedure entirely a simple and safe one, as a published perforation rate of 2 per cent and a mortality rate of 1.5 per cent from instrumentation discloses. Again, the removal of tissue for pathologic examination requires skill to avoid hemorrhage and perforation. A section should be taken through the rim of the ulcer without maceration, and in sufficient size. Pathologists are handicapped considerably, and led into diagnostic error, if poor material is submitted.

Ulcers as seen through the esophagoscope may present certain distinguishing characteristics. Thus peptic ulcer typically is flat, clean-cut, with a granular bleeding base, and is surrounded by a zone of hyperemia. Tuberculous ulcer is whitish or grayish, irregular in outline with a somewhat moth-eaten aspect. Whitish or yellowish granules may be seen in the walls and base. Syphilis does not always present a characteristic punched-out ulcer, but may be suspected by an adjacent mucous patch or gumma. Carcinoma in contradistinction displays infiltration, fungation, or both. In any case the endoscopic appearance must be evaluated in terms of manifestations of disease elsewhere in the body. The final diagnosis therefore will be based upon the local appearance of the ulcer in conjunction with the clinical picture and tissue examination.

Treatment.—The treatment of benign ulcers of the esophagus must be carried out in three directions: first, the general condition of the patient; second, against any specific etiologic factor, such as syphilis; and last, the ulcer itself.

1. *Treatment of General Condition.*—Supportive tonic treatment is important. Systemic disease and associated disease should be cared for. Foci of infection should be eliminated especially in the mouth, throat, and upper respiratory tract.

2. *Local Treatment of the Ulcer.*—Many benign ulcers, particularly the simple types that are associated with esophagitis or mild trauma, heal spontaneously, without any special therapeusis other than the taking of a bland diet. There is little to be looked for in specific therapy. For the very rare tuberculous ulceration, 50 per cent lactic acid applied locally has met with some palliative success. The healing of ulcers of all types may be hastened by the swallowing of bismuth or barium in powder or paste, and by the application through the esophagoscope to the ulcer of 10 per cent silver nitrate or chlorlyptus oil as practiced by Dr. Ellen J. Patterson. Where pain is intense, orthoform or anesthesin in ½ per cent solution or ethyl aminobenzoate lozenges, as suggested by Vinson and Butt, may mitigate the severity.

3. *Treatment of Stenosis and Spasm.*—Both will require mechanical dilatation with bougies or bags. Administration of antispasmodics such as bromides and belladonna for spasm has proved disappointing. Where spasm persists stubbornly in spite of faithful bouginage, one should seek a reflex cause elsewhere. Any intraabdominal lesion that may be a reasonably predisposing or aggravating factor should be corrected. As in the case we are reporting, the spasm factor may be difficult to overcome. If one believes with Sauerbruch[9] and Jackson that spasm at the lower end of the esophagus is not inherent in the esophageal musculature but is in reality a phrenospasm, and as Jackson puts it, "a pinch-cock action of the diaphragm," then phrenic nerve interruption should relieve the spasm. Sauerbruch noted that the usual resistance felt at the cardia by the finger through a gastrostomy opening disappeared after a bilateral phrenic nerve section. Phrenicectomy has been tried in so-called cardiospasm or preventiculosis with indifferent results, perhaps because bilateral phrenic paralysis has not usually been practiced. Vagus nerve section has also been performed and found ineffective.

4. *Operative Surgery.*—(a) *Gastrostomy* will be needed to rest the esophagus, and for feeding purposes in severe and stubborn cases of ulceration, particularly in those that have excessive spasm and stenosis, if an indwelling tube is not feasible, and also, if retrograde dilatation is to be carried out. Hurst[10] states if an ulcer is not healed within a year a gastrostomy is indicated. This seems to us too long to wait. The operation usually causes no detriment to the patient's welfare if the stoma is properly constructed. The question may be raised, however, as we are doing in our own reported case, as to whether a long continued gastrostomy may not be conducive to the production or maintenance of spasm, and thus defeat other therapeutic measures undertaken for its relief.

(b) *Esophagectomy* will very rarely be indicated for benign ulceration. In view of the magnitude and seriousness of the operation it should be a procedure of last resort for intractable ulceration that borders on, and threatens to become malignant. Eggers last year reported one case of successful resection of the terminal esophagus in which the biopsy report was carcinoma, but when the tumor was examined the ulceration was found to be nonmalignant.

(c) *Diverticulectomy* should be performed particularly in cases in which ulcers can be demonstrated because of their proneness to perforation. In the thoracic esophagus this operation might easily not be feasible, and call for an esophagectomy as the alternative.

(d) *Esophagoplasty* will be needed to restore the food canal in patients who have impermeable or almost closed stenosis which does not respond to dilatation.

(e) *Perforations.* In the cervical region the problem is relatively simple. Early incision and packing off of the thoracic inlet after the manner of Marschik will be successful largely to the degree that mediastinitis is prevented or controlled. In perforations of the thoracic esophagus the habit has been to wait development of localized abscess formation before undertaking surgery. In the great majority of acute perforations, however, mediastinitis will be fulminating and speedily fatal, so that there is nothing to gain and everything to lose by delay. A posterior mediastinotomy should be done at once at the appropriate level in the thorax, and the area of the perforation packed and drained. I believe that early operation will save some lives that would be sacrificed by watchful waiting, while the converse is not true. Perforations into the pleura and lung may be handled more leisurely.

The prognosis in cases of perforated ulcer will further depend upon the well-established factors of the virulence of the organism, the responding and resisting power of the individual, and the freedom from complicating or constitutional disease.

CONCLUSIONS

A review of this subject of benign ulceration of the esophagus leads one to believe:

1. That ulcers occur much more frequently than they either are diagnosed or can be diagnosed.

2. Esophagoscopy should be utilized freely in the examination of patients with gastric symptoms, and uniformly in those in whom symptoms persist in spite of rational treatment.

3. Biopsy in addition to endoscopy will often be needed to establish the diagnosis.

4. Whereas many ulcers seem trivial and innocuous, they should all be treated carefully and seriously in view of the tragedy and disaster that follow in the train of complicating hemorrhage, perforation, and stenosis.

5. Whereas most benign ulcers respond quickly to treatment, a few do not, and even though healed lead to persistent disturbance of esophageal function. These cases will test the ingenuity and patience of the clinician.

REFERENCES

1. Jackson, Chevalier: J. A. M. A. **92:** 369, 1929.
2. Stewart, M. J.: J. Path. & Bact. **132:** 917, 1929.
3. Bartels, E. C.: Arch. Path. **20:** 369, 1935.
4. Lockard, L. B.: Esophageal Tuberculosis, Laryngoscope **23:** 561, 1913.
5. Friedenwald, J.: Am. J. M. Sc. **177:** 1, 1929.
6. Tileston, W.: Am. J. M. Sc. **132:** 240, 1906.
7. Guisez, J.: J. Laryngol. & Otol. **40:** 141, 1924.
8. Cushing, Harvey: Surg. Gynec. Obst. **55:** 1, 1932.
9. Sauerbruch, F., and Haecker, F.: Zur Frage der Cardiaverschluss der Speisnöhre, Deutsche med. Wchnschr. Aug. 2, 1903.
10. Hurst, A. F.: J. A. M. A. **102:** 582, 1934.

SURGICAL MANAGEMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA*

Report of Two Cases

N. Logan Leven, M.D.†
St. Paul, Minnesota

THOMAS GIBSON in 1696 first observed a case of congenital atresia of the esophagus with a tracheo-esophageal fistula, noting the typical feeding difficulties and accurately describing the postmortem findings. Mackenzie, Griffith and Lavenson, Cautley, Plass, Phelps and Rosenthal have carefully reviewed the literature and collected all recorded cases of congenital anomalies of the esophagus. (Fig. 1.) In 1931 Rosenthal collected 255 cases of congenital atresia of the esophagus and found that 205 had associated tracheo-esophageal fistulas.

In this most common type of atresia of the esophagus (Fig. 2) the upper segment terminates blindly just above the bifurcation of the trachea, while the lower segment has a fistulous communication with the trachea usually about 0.5 to 1.0 cm. above the bifurcation or more rarely with the bronchus. The upper culdesac is usually hypertrophied and dilated and has an average length of 3 to 4 cm. The lower segment of the esophagus at the cardiac end is usually of normal size but often diminishes in caliber toward its tracheal opening.[16]

The symptomatology associated with this anomaly is so characteristic that it should be readily recognized. At birth the child appears to be well nourished and usually well developed but has difficulty with large amounts of frothy mucus filling mouth and pharynx, and drooling from the side of the mouth. When fed, the child eagerly takes the breast and after a few swallows stops, ceases to breathe, becomes cyanotic, and regurgitates frothy mucus and feedings through the nose and the mouth. The child appears as if it would drown, but after a period of lifeless relaxation usually recovers and repeats this episode with each subsequent feeding. These infants rapidly lose weight due to starvation and dehydration and often develop an aspiration pneumonia. The average weight loss before death according to Brenneman[2, 3] is about 25 to 40 per cent.

Physical examination as a rule reveals only frothy mucus in the pharynx, signs of dehydration, and frequently distention of the up-

*Read (by invitation) at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.
†From the Department of Surgery, University of Minnesota.

per abdomen due to air in the stomach.[2, 3] Rosenthal states that other congenital anomalies are frequently present but usually are not incompatible with life.

The diagnosis of atresia of the esophagus is made on the typical history, obstruction to the passage of a catheter or bougie at 10 to 12 cm. from the alveolar margins, and x-ray visualization of the blind pouch with a barium meal or by esophagoscopic examination. The

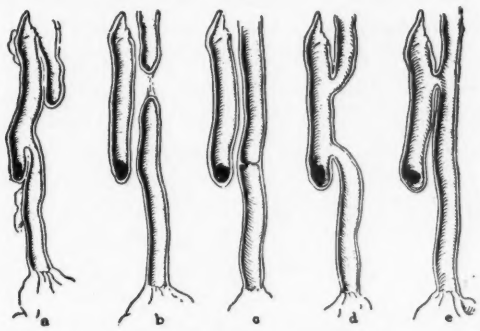


Fig. 1.—Types of congenital deformity of the esophagus (after Vinson).

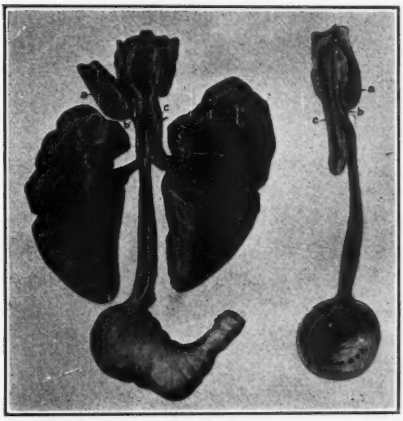


Fig. 2.—Commonest type of congenital atresia of esophagus with tracheo-esophageal fistula.
a, Blind pharyngeal pouch.
b, Pedicle connecting pouch to esophagus (not always found).
c, Opening of esophagus into the trachea. (After Broyles.)

presence of air in the stomach in association with atresia of the esophagus is evidence of a fistulous communication with the lung.[17] This fistula may be visualized and catheterized at bronchoscopic examination as suggested by Tucker and Pendergrass.

The prognosis in these cases has usually been considered hopeless. The longest survival is the case reported by Schmidgall in which

fluids were given per rectum after twelve days, a gastrostomy was performed at twenty days, and the child lived until twenty-eight days of age. As a rule these children die within the first week. In cases of simple atresia of the esophagus gastrostomy should be sufficient. Abel in a case of obstruction by a membranous diaphragm obtained a cure by rupture of the membrane during esophagoscopy. The common type of atresia of the esophagus, however, presents three problems: (1) feeding; (2) management of the fistulous communication of the lower segment of the esophagus with the trachea; and (3) care of the blind pouch of the upper esophageal segment.

For purpose of feeding, the most frequent procedure used is gastrostomy.[6, 18, 23, 25, 28] This of itself may hasten death, since fluids injected through the gastrostomy tube may be promptly regurgitated through the fistula into the trachea resulting in an aspiration pneumonia. To avoid this, jejunostomy has been suggested and tried but is ineffectual[2, 6, 11, 17, 26] since gastric contents may still be regurgitated into the lung. By placing a catheter in the lower esophagus and applying suction to it, in addition to a gastrostomy tube for feeding, Wangensteen attempted to prevent regurgitation of gastric contents into the lung. In spite of this, apparently some regurgitation took place and death by bronchopneumonia occurred. In one case I have employed a jejunostomy tube for feeding combined with a gastrostomy tube for evacuation of the stomach, with no success.

Ligation of the esophagus at the cardia has been combined with gastrostomy to prevent regurgitation of gastric contents into the lung.[21, 24, 27] Villemin in 1904 suggested division of the cardiac end of the esophagus, ligating it and bringing the cardiac end of the stomach into the wound forming a cardiostomy for feeding. By these procedures a blind pouch of esophagus is formed in which secretions will collect, become infected, and empty into the lungs. There is also a definite danger of the occluding ligature cutting through the wall of the esophagus. Intrathoracic operations directly attacking the fistula have been devised for these cases. Division and closure of the fistula at its junction with the trachea together with connection of the esophageal segments by a tube[12] or anastomosis of the segments by a modified Murphy button[9] or exteriorization of the lower segment making a dorsal esophagostomy[10] for feeding purposes have all been suggested. Richter in two cases performed a gastrostomy for feeding after ligating the upper portion of the distal segment of the esophagus at its junction with the trachea. Neither case was successful. Although these procedures take care of the fistula, their mortality would prohibit their use in these tiny infants.

It is the censensus of opinion that the blind pouch of the upper esophageal segment may best be handled by a cervical esophagos-

tomy. I have learned, however, that these patients if not fed by mouth will do quite well with intermittent aspiration of mucus and saliva from the mouth with a bulb syringe. This stage of the operative management may therefore be deferred.

To circumvent some of the difficulties in treating these cases, I have carried out a procedure which attacks the fistula indirectly.

DESCRIPTION OF OPERATION

After adequate preoperative preparation by administration of subcutaneous fluids if dehydration is present, the operation is carried out under local anesthesia. The abdomen is opened through an upper left rectus incision which extends up to the rib margin. (Fig. 3.) A small

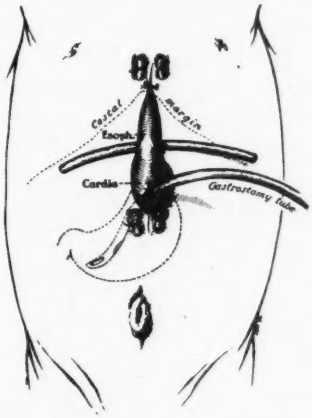


Fig. 3.—First stage operation showing exteriorized cardiac end of esophagus and stomach.

amount of clear amber-colored fluid is often noted on opening the peritoneum. The liver, which is relatively very large in these newborn infants, presents in the wound and makes the exposure a little more difficult.[6, 18, 25] The stomach is gradually retracted and packed off until the cardia end is reached. Using blunt dissection with a curved forceps, the subdiaphragmatic esophagus and the cardiac end of the stomach are mobilized. A rubber tissue drain is then passed under the mobilized esophagus and used for traction. By blunt dissection a centimeter of the mediastinal esophagus can be pulled into the abdominal cavity. By depressing the abdominal wall and exerting moderate traction on the rubber tissue drain the cardiac end of the esophagus and stomach can readily be brought up into the wound. Two mattress sutures of chromic catgut bring peritoneum and rectus sheath under the exteriorized cardiac end of stomach and esophagus.

A multiple purse string type of gastrostomy is then made in the stomach distal to the exteriorized portion. The upper and lower ends of the wound are closed with mattress sutures through the muscle and peritoneum. The skin is closed with vertical mattress sutures tied over rubber tubing. A soft rubber catheter is placed under the exteriorized segment, and the ends are fastened to the abdominal wall with adhesive tape. An angulation is thus formed at the cardia and proximal to the gastrostomy, which will effectually prevent regurgitation of gastric contents into the lungs so that an adequate diet can be fed through the gastrostomy tube immediately. This first stage is followed by very little postoperation reaction.

Because of leakage about the gastrostomy tube and perforations which occur in the exteriorized segment of stomach and esophagus, it is advisable to cut across this portion of stomach in two to three weeks and reconstruct the gastrostomy.

A cervical esophagostomy would be done as the second stage procedure, and at a distant future date an antethoracic esophagoplasty could be carried out to establish continuity of the gastrointestinal tract.

It probably would be unnecessary to do anything about the external fistula of the subdiaphragmatic esophagus, since the small amount of mucous drainage should not be of great inconvenience.

REPORT OF CASES

CASE 1.—Hospital No. 635670. Male infant, four days old, admitted to the University Hospital Feb. 17, 1935. Weight at birth was 3,900 grams. During the first twenty-four hours after delivery the chief symptom was regurgitation of mucus. When given the breast at twenty-four hours there was prompt regurgitation of mucus and milk accompanied by an attack of cyanosis and choking. Difficulty with mucus was controlled by postural drainage and aspiration. Two hundred and fifty cubic centimeters of saline had been injected into the peritoneal cavity on the fourth day, prior to admission to the hospital.

Examination showed a well-developed, well-nourished male infant weighing 3,300 grams. Temperature was 102.4° F. An excess of frothy mucus was present in the mouth and pharynx. Dullness to percussion was present in the right upper chest. The remainder of the physical examination showed no other evidence of abnormalities. Passage of a catheter met obstruction 12 cm. from the alveolar margin. Injection of a small amount of barium demonstrated that the upper segment of the esophagus ended in a blind pouch. The barium was aspirated from the esophagus after x-ray examination to prevent aspiration into the lungs.

An atelectasis of the upper lobe of the right lung and a large amount of gas in the gastrointestinal tract were also noted on x-ray examination (Fig. 4). After administration of fluids subcutaneously the previously described operation was performed under local anesthesia February 18. The operation was well tolerated; the infant gained 400 grams in weight, and the atelectasis of the right lung cleared during the following week (Fig. 5). A nasal suction catheter was inserted to keep the upper pouch of the esophagus free from mucus. At the end of one week considerable leakage developed about the gastrostomy tube. The feeding catheter was passed through the pylorus into the duodenum as suggested by Villemin, and, although gastric juice continued to leak about the tube, the feedings introduced di-

rectly into the duodenum were well retained. At three weeks a bilateral otitis media developed, and both ear drums were opened. After a period of improvement, the otitis media recurred at eight weeks of age. Bronchopneumonia then developed and the child died three days later, May 21, at ninety-eight days of age. Weight at the time of death was 4,290 grams.

Postmortem examination showed an extensive bilateral bronchopneumonia. An atresia of the esophagus was present, the upper segment ending blindly just above the bifurcation of the trachea and the lower esophagus communicating with the trachea about one centimeter above the bifurcation. The wall of the upper esophageal segment was markedly hypertrophied and the caliber about twice that of normal esophagus.

The peritoneal cavity was free from infection. No other anomalies of development were found.

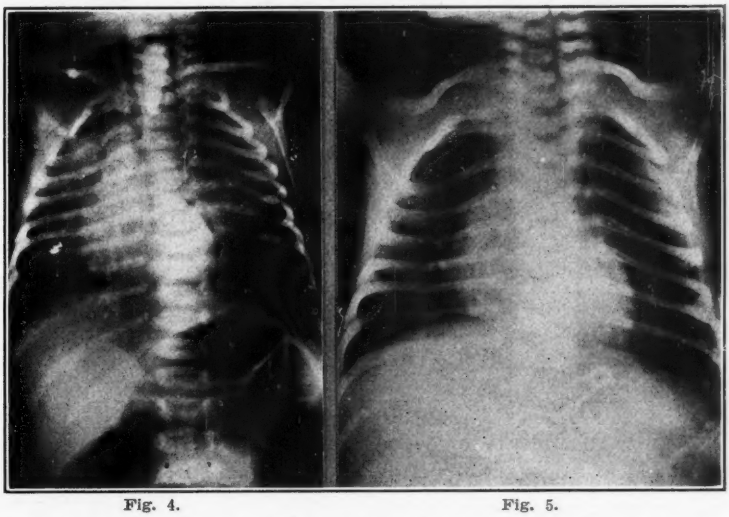


Fig. 4. Fig. 5.

Fig. 4.—Roentgenogram of Case 1 on admission, showing blind pouch of upper segment of esophagus, air in gastrointestinal tract, and atelectasis of right upper lobe of lung.

Fig. 5.—Roentgenogram of Case 1, showing improvement of atelectasis of right lung.

CASE 2.—Hospital No. 647262. Male infant admitted to the University Hospital March 24, 1936. Born at the Minneapolis General Hospital March 22, 1936, about four weeks premature, weighing 1,910 grams. Typical history of attacks of regurgitation, choking and cyanosis when feedings were given. A diagnosis of congenital atresia of the esophagus with tracheo-esophageal fistula was confirmed by obstruction to the passage of a catheter, visualization by x-ray of the upper segment outlined by barium, and the presence of air in the gastrointestinal tract. Although subcutaneous fluids had been given, the weight on admission to the University Hospital was 1,780 grams. On March 25 under local anesthesia the cardiac end of the esophagus and stomach were exteriorized and a gastrostomy was performed as in Case 1.

Intermittent aspiration of mucus from the mouth is being used in this case instead of an indwelling nasal suction catheter in the blind pouch of the upper

segment of esophagus. By this means it is hoped to avoid the danger of otitis media from irritation by a nasal tube.

Similarly in this case leakage and prolapse of gastric mucous membrane occurred about the gastrostomy necessitating the passage of the feeding tube through the pylorus into the duodenum.

At four weeks of age, the exteriorized portion of stomach was divided and the gastrostomy reconstructed. While the stoma was improved, some prolapse of gastric mucous membrane recurred which will require further attention.

In view of the fact that intermittent aspiration with a bulb syringe of mucus from the mouth has been very effectual, the cervical esophagostomy need not be considered an urgent procedure.

This infant is now forty-two days old and weighs 2,000 grams.*

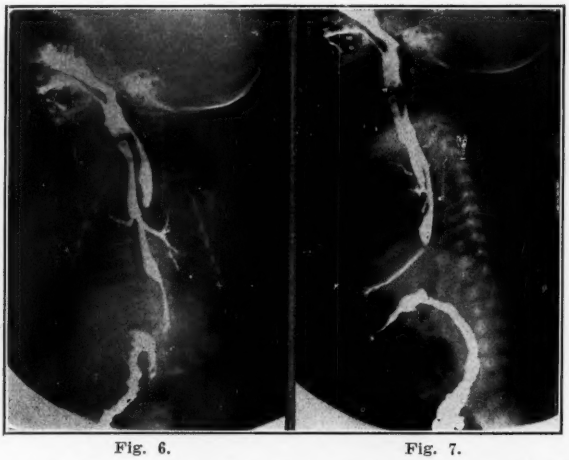


Fig. 6. Fig. 7.

Figs. 6 and 7.—Postmortem roentgenograms, Case 2, after lipiodol filling of the esophagus and trachea demonstrating the upper blind pouch of the esophagus, the tracheo-esophageal fistula and the exteriorized cardiac ends of esophagus and stomach.

COMMENT

Experimentally in a study of intestinal obstruction Wangensteen and Chunn have found that dogs with ligation of the cervical esophagus show findings similar to cases of congenital atresia of the esophagus. Large amounts of frothy mucus drool from the mouth, and, when attempt is made to swallow water, a severe attack of choking ensues. These dogs died in two days.

In the first case reported in this paper an attempt was made to keep the blind pouch of the upper esophageal segment empty of mucus to prevent aspiration pneumonia. The indwelling nasal catheter used was effectual but probably was also a predisposing factor in the development

*This infant, Case 2, died May 16, 1936, at fifty-three days of age after one week of elevated temperature of 100° to 102.4°. Weight at the time of death was 2,000 grams. Postmortem examination demonstrated the congenital atresia of the esophagus with a fistula into the trachea just above the bifurcation. The foramen ovale showed a two millimeter opening. No evidence of infection was found in the lungs, pleurae, or peritoneal cavities. (See Figs. 6 and 7.)

of otitis media. Peyton in a study of developmental anatomy of the head has shown that the eustachian tube in the newborn infant is horizontal or slightly inclined toward the middle ear while in the adult it presents a sharp inclination from the middle ear to the nasal cavity (Fig. 8). Thus in an infant infection could extend from the nasal cavity to the middle ear much more readily than in adults. This may account for the prompt development of otitis media in this case from infection by irritation of the indwelling catheter. In adults otitis media is extremely rare in association with prolonged use of a nasal suction catheter.

In cases of congenital atresia of the esophagus with associated tracheo-esophageal fistula the usual methods of gastrostomy, jejunostomy, or combinations of these have proved unsuccessful in preventing regurgitation of gastric contents into the lung causing an aspiration pneumonia. The addition of ligature at the cardiac end of the

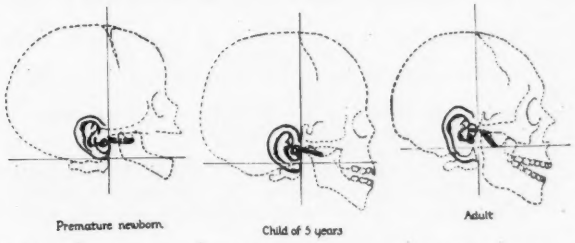


Fig. 8.—The growth changes occurring in the eustachian tube showing position in newborn, at five years, and in the adult (after Peyton).

esophagus which would effectually take care of the communication with the lung carries the danger of forming an obstructed segment of esophagus, the contents of which may become infected, and empty into the lung. Intrathoracic operations would probably carry too great a mortality in these infants to be advisable.

By the method presented here regurgitation of gastric contents into the lung is prevented first by angulation of the cardiac end of the esophagus and stomach and later by division across the cardiac end of the stomach. By gastrostomy adequate feedings may then safely be given. It is shown that the danger of aspiration of mucus from the mouth into the trachea can temporarily be taken care of by intermittent aspiration of the mouth with a bulb syringe, making the cervical esophagostomy no emergency procedure.

CONCLUSION

A new method of surgical management of cases of congenital atresia of the esophagus with tracheo-esophageal fistulas is described. The

cardiac end of the esophagus and the stomach are mobilized and exteriorized. Feedings through a gastrostomy tube in the stomach distal to the exteriorized segment are thus prevented from regurgitating into the lung by the angulation formed in the cardiac end of the esophagus by the operation.

A cervical esophagostomy and later an antethoracic esophagoplasty would be required to reestablish the continuity of the gastrointestinal tract.

Two cases are reported in which the described procedure was used. The first child died at ninety-eight days of age, while the second infant is still alive and is forty-two days old.

REFERENCES

1. Abel, A. Lawrence: Oesophageal Obstruction, London, 1929, pp. 54-56, Oxford Medical Publications.
2. Brenneman, J.: Congenital Atresia of the Esophagus, With Report of Three Cases, Am. J. Dis. Child. **5**: 143, 1913.
3. Brenneman, J.: Congenital Atresia of the Esophagus, Am. J. Dis. Child. **16**: 143, 1918.
4. Broyles, E. N.: Oesophagus, p. 1, Practice of Surgery, Dean Lewis, Hagerstown, Md., 1933, IV, Chap. 8, W. F. Prior Co., Inc.
5. Cautley, Edmund: Malformation of the Esophagus, Brit. J. Child. Dis. **14**: 1, 1917.
6. Demoulin, M. A.: Imperforation de l'oesophage chez un enfant nouveau-né. Bull. et Mém. Soc. de chir. de Paris **30**: 730, 1904.
7. Gibson, Thomas: Quoted by Plass.
8. Griffith, J. P. C., and Lavenson, R. S.: Congenital Malformation of the Esophagus, With the Report of a Case, Arch. Ped. **26**: 161, 1909.
9. von Hacker, V.: Neue Deutsche Chirurgie, Stuttgart, 1926, 34 Band S. 53, F. Enke.
10. Iglauer, S.: A Proposed Operation for the Relief of Congenital Atresia of the Esophagus, Ann. Otol. Rhin. & Laryng. **43**: 1147, 1934.
11. Jones, E. O., and Manning, J. B.: Congenital Atresia of the Esophagus, J. A. M. A. **6**: 810, 1916.
12. Lilienthal, H.: Thoracic Surgery, Philadelphia, 1925, p. 321, W. B. Saunders Co.
13. Mackenzie, Morell: A Manual of Diseases of the Throat and Nose, v. 2, New York, 1884, pp. 149-158, Wm. Wood & Co.
14. Peyton, W. T.: Unpublished data.
15. Phelps, K. A.: Congenital Anomalies of the Esophagus, Ann. Otol. Rhin. & Laryng. **39**: 364, 1930.
16. Plass, E. D.: Congenital Atresia of the Esophagus With Tracheo-Esophageal Fistula, Johns Hopkins Hosp. Rep. **18**: 259, 1919.
17. Reid, M. R.: An Aid in the Diagnosis of a Certain Type of Congenital Esophageal Stenosis, J. Pediatrics **1**: 87, 1932.
18. Renault, J., and Sebilleau, J.: Malformation congenitale de l'oesophage (oblitération oesophagienne et communication oesophago-trachéale), Arch. de Med. des Enfants **7**: 630, 1904.
19. Richter, H. M.: Congenital Atresia of the Oesophagus: An Operation Designed for Its Cure, Surg. Gynec. Obst. **17**: 397, 1913.
20. Rosenthal, A. H.: Congenital Atresia of the Esophagus With Tracheo-esophageal Fistula, Arch. Path. **12**: 756, 1931.
21. Sarnoff: Quoted by Iglauer.
22. Schmidgall, G.: Angeborene Atresia des Oesophagus und Oesophagustracheal-fistel, Arch. f. Kinderh. **64**: 72, 1915.
23. Sedillot: Quoted by Mackenzie.
24. Smith, E. D.: The Treatment of Congenital Atresia of the Esophagus, Ann. Surg. **37**: 157, 1923.
25. Steele, C.: Case of Deficient Oesophagus, Lancet, No. 2, p. 764, 1888.
26. Strong, R. A., and Cummins, H.: Congenital Atresia of the Esophagus With Tracheo-esophageal Fistula, Am. J. Dis. Child. **47**: 1299, 1934.

27. Tucker, G., and Pendergrass, E. P.: Congenital Atresia of the Esophagus: A New Diagnostic Technique, J. A. M. A. 101: 1726, 1933.
28. Villemin: Imperforation de l'oesophage, Arch. de Med. des Enfants 7: 511, 1904.
29. Vinson, P. P.: Congenital Strictures of the Esophagus, J. A. M. A. 80: 1754, 1923.
30. Wangensteen, O. H.: Personal communication.
31. Wangensteen, O. H., and Chunn, S. S.: Studies in Intestinal Obstruction, III. Simple Obstruction, Arch. Surg. 16: 1242, 1928.

DISCUSSION ON "THE MUSCULAR COAT OF THE ESOPHAGUS AND ITS DEFECTS," BY LERCHE; "DIAGNOSIS AND TREATMENT OF BENIGN ULCERS OF THE ESOPHAGUS, WITH CASE REPORT," BY DECKER; AND "SURGICAL MANAGEMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA," BY LEVEN

DR. FRANZ TOREK.—Dr. Lerche has pointed out that the study of the anatomy should always be the basis of many of our conclusions. The same is true of physiology. And in that respect, in reference to Dr. Decker's paper, it should be remembered that the mucosa of the esophagus is not immune to the acid secretion of the stomach. Consequently we have pretty well adopted it as a fact that when there is a peptic ulcer at the lower end of the esophagus, some of the acid of the gastric juice has been forced into the esophagus.

This consideration naturally would lead us to consider it in the treatment. If there is a peptic ulcer in the lower end of the esophagus that has not yet advanced so far as to be a chronic ulcer, with a firm base, it ought to be possible to heal it without its being directly attacked by the esophagoscope. That has been done.

In the first place we should find out what is the cause of regurgitation. One of the causes is the presence of a simultaneous lesion at the pylorus or duodenum. The coexistence of lesions at the lower end of the esophagus and at the pylorus, especially cardiospasm and pylorospasm, has been reported by Lehman, Cameron, Bolan and Zusch even in infants and children, not only in adults.* So we might draw the conclusion that the spasm which is brought about at the pylorus, in consequence of the pyloric or duodenal ulcer, is often responsible for throwing the gastric juice up into the esophagus and, if that occurs often enough, producing a peptic ulcer. Consequently our treatment may have to be directed to the presence or supposed presence of some lesion at the pylorus. I say presence or supposed presence because in the early beginnings of a peptic or duodenal ulcer we may have no way by x-ray examination to say positively there is a lesion. There may be a lesion, but too small to be shown.

I understand that Dr. Chevalier Jackson, Sr., has treated some of his cases (of course he has seen a great many of the peptic ulcers) by the introduction of the duodenal tube. The duodenal tube is carried down into the stomach and should also be carried down into the duodenum, especially because its presence in the duodenum would probably to a great extent counteract any spasm at the pylorus, besides leaving the ulcers both in the esophagus and in the duodenum free from any irritation by food. If Dr. Jackson is present, I hope he will tell us something about his experience.

In the more advanced cases with chronic ulcers and thick walls, we probably would not accomplish very much without the aid of a gastrostomy. I have had such a case, with enormous dilatation of the esophagus, cardiotonic regurgitation of decomposed food, and copious bleeding from the ulcers of the esophagus. The patient was very weak from lack of food, as most of it was regurgitated, and from loss of

*Lehman, W.: Die Aetiologie der sog. spastischen Erkrankungen des Magen-Darm-Kanals, Beitr. z. klin. Chir. 151: 395, 1931.

blood. In that case I did a gastrostomy by which he was promptly relieved. Later, the ulcers were treated locally by Dr. Jackson, with the aid of the esophagoscope. The patient was cured; his stomach fistula remained open about a year. It closed by itself after the ulcers were healed.

DR. PHILEMON E. TRUESDALE.—These three papers on the esophagus have been, it seems to me, very important and most enlightening, particularly that on minute anatomy of the esophagus, also the paper by Dr. Decker, dealing with ulcerations at the lower end of the esophagus. We became interested in that subject on account of the repeated references to cardiospasm, with hypertrophy of the cardiac sphincter, and the irritability of the cardiac sphincter at the lower end of the esophagus.

We had some skepticism about the existence of a sphincter at the lower end of the esophagus, and therefore we made microscopic section examinations of the lower end of the esophagus in ten specimens removed postmortem. In no one of the specimens could we find a collection of fibers at the lower end of the esophagus that could be called a cardiac sphincter.

In the course of that examination, we found that there were islands of gastric mucosa, 2, 3, and 4 cm., above the junction of the esophagus and the stomach, and it seemed very likely that the ulcers which occur at the lower end of the esophagus occur where there is gastric mucosa, gastric glands, and that the ulcers which subsequently become carcinomatous, probably, are adenocarcinomatous, having their origin in the stomach tissue instead of the esophageal mucosa.

We know that carcinoma of the esophagus, other than in the lower part, is quite rare. A great majority of the carcinomas of the esophagus are at the lower end, and it does seem probable that, in view of the fact that these little islands of gastric mucosa exist in the lower end of the esophagus, many if not all of these cancers develop on gastric mucosa.

DR. HOWARD LILIENTHAL.—There is an operation which has been described first by me and afterward by Sauerbruch in his last edition, which I have called esophagogastrostomy. In these desperate cases, in which nothing else seems to be possible, I have advised this operation for carcinoma in the lower part of the esophagus. The procedure might be advisable in cases which are not carcinoma but in which the outlook is otherwise hopeless. I did the operation in one case and lost the patient five days afterward, a carcinoma case, from pneumonia. The operation itself is not difficult. It is posterior, mediastinal esophagogastrostomy. Those of you who are interested can find the method described in a book that I wrote about ten or eleven years ago.* I recommend the procedure very strongly, and shall certainly use it myself in case I encounter a condition of this kind. It is decidedly better than mere gastrostomy, because there is no external opening and the patient swallows normally. The patient on whom I did operate, for four days swallowed not only fluids but soft solids, and without any distress. At the postmortem (following pneumonia) there was no leakage discoverable.

DR. C. L. JACKSON.—Dr. Decker's paper is very interesting to me. It is a very complete review of this whole subject, but I should like to mention one point which has a bearing on diagnosis and probably on etiology.

We have found that in a great many of the cases of "peptic ulcer" of the esophagus, there is some degree of herniation of the stomach, sometimes a very slight herniation, difficult to pick up, especially when the roentgen examination is made only in the upright posture. In some of the cases the condition has seemed to be one of congenitally short esophagus, with supradiaphragmatic stomach rather than herniation.

*Thoracic Surgery, Saunders, 1925.

With regard to treatment, these patients are benefited by sleeping with the head and shoulders elevated, because retention of the gastric secretions in the supradiaphragmatic stomach is thus avoided. Patients with a herniation not sufficient to justify surgical procedures will be greatly benefited by adding this measure to the other conservative measures outlined by Dr. Decker.

Dr. Torek spoke of the treatment of these peptic ulcers of the esophagus with the duodenal feeding tube. We have used this method only in one or two cases. The association of gastric and duodenal ulcers with ulcers in the lower end of the esophagus is a very interesting thing. I reported a few cases of such associated lesions at the meeting of the American Therapeutic Society in Baltimore in 1932, and I am interested in hearing that Dr. Decker has also seen some.

DR. POL N. CORYLLOS.—I wish to join you in congratulating Drs. Lerche, Decker and Leven for their excellent papers on esophagus.

We all admired the scholarly description of the anatomy of the esophagus given by Dr. Lerche. I believe, however, that, without denying the importance of the anatomical construction and physiologic function of the muscularis of the esophagus in the pathogenesis of cardiospasm, we must admit that it is limited to its initial phases only. In fact, cardiospasm may be of reflex origin or it may be due to dissociation of the action of sympathetic and parasympathetic innervations; most frequently, however, it is due to inflammatory lesions such as acute or subacute esophagitis or ulcerative lesions as the ones described by Dr. Decker. It is different for chronic cardiospasm of long duration. In these cases, whatever the etiology may have been, the pathology consists, as a rule, of fibrotic thickening of the muscular layer of the lower third of the esophagus. MacMillan and Mosher have shown this fact very clearly. The spastic element is of secondary importance, and the diaphragmatic pinch-cock action of Ch. Jackson or spasm of the cardiac sphincter, if present, are of little importance in the production of permanent esophageal stenosis.

This being the case, I wonder why the operation proposed by Heller in 1913 under the name of esophagectomy is so little used in this country, in cases of cardiospasm which cannot be relieved by forcible dilation. A considerable number of these operations have been performed in Germany, Russia, France; lately Scrimger of Montreal reported three cases.

I had the opportunity to operate in 1934 upon a white girl of fifteen years for an intractable cardiospasm, whose case had been followed by others and by me since the child was eleven years of age. The onset of the disease was quite sudden and progressed rapidly. Esophagoscopy in 1931 showed the presence of two small ulcerations of the esophageal mucosa 1 cm. above the cardia. In 1932 these ulcerations were healed and could not be seen with the esophagoscope. Repeated forcible dilations with the Plummer esophageal dilator were made. At the beginning these dilations improved her condition for periods of one to two months; gradually there was little and finally no improvement at all. During the three months preceding operation no improvement whatever was brought about even by dilations performed under general anesthesia. The patient was rapidly losing weight and going downhill.

My operative plan was to attack the esophagus by abdominal approach and to perform Heller's operation. If this could not have been done, I would have tried esophagogastrostomy by abdominal approach according to the procedures of Keller and Churchill. Were these operations not feasible I would have closed the abdomen and proceeded later by the thoracic approach.

Under spinal anesthesia a median upper abdominal incision was made. The esophagus was gently dissected from its covering, and the vagi were retracted. The esophagus was dissected free from the crura of the diaphragm and gradually

lowered into the abdomen until the upper limits of its constricted part and the beginning of its dilated portion became visible. The narrowed portion of the esophagus was the size of an index finger; it was hard on palpation and resistant in consistency. The length of this segment from the cardia to the dilated portion was 9 cm. With great care and using a very sharp blade, the muscularis of the cardia was incised until the mucous became visible. Then the mucosa was gradually separated from the muscularis with the handle of the knife; the muscularis was then sectioned little by little until the ring limiting the retracted from the dilated portions was reached. The incision of the muscularis gradually opened up, and the mucosa herniated through it. No ligatures were necessary except on two small bleeders. No sutures were placed in the muscularis, and the abdominal wound was sutured in three layers with a small cigarette drain left in the wound; this was removed twenty-four hours later.

The postoperative course was strikingly simple. Twelve hours after operation the patient was given fluids; three days later she was put on soft diet, and on the fifth postoperative day solid food was given. It was remarkable that during the operation when the incision of the muscularis was completed the patient said that she felt great relief. This patient gained 30 pounds within four months following operation.

I thought it worth while to report this case because it shows that the operation of Heller, which is a relatively simple and harmless procedure, can be of great help in these cases; especially that it should be better known and at least tried before much more dangerous operations, such as transpleural gastroesophagostomy, are decided upon.

DR. JOHN V. BOHRER.—I should like to mention Dr. Leven's case and compliment him on the unique way in which he controlled the regurgitation of food. Most of these cases have been operated on by a simple gastrostomy, with evil results in that they regurgitate. In the literature one finds a number of cases in which gastrostomy has been rather successful; that is, the patient would live for quite a number of days, which simply means that the fistulous tract either was absent or was represented only by fibrous bands, and there actually was no fistula into the trachea. I attempted to solve this problem by making a direct attack upon the fistulous area, where it united with the trachea.

We have had a number of these cases on the Children's Surgical Service, Bellevue Hospital. Eighty per cent of them fall in the same type of congenital deformity as the case Dr. Leven has reported; that is, where the fistulous communication is at the bifurcation of the trachea. Our last case was admitted about one year ago. It was a premature child and had other congenital deformities, which is a common finding. I realized that it probably would not survive an operative procedure, but without operation it was hopeless, and I wished to demonstrate the feasibility of a direct attack in attempting to overcome the problem.

The blind pouch representing the upper portion of the esophagus must be kept empty by frequent aspirations until it is apropos to do an esophagotomy and make a skin fistula of that portion of the esophagus.

My idea was to sever the esophagus from the trachea at the fistulous junction, closing the tracheal end by ligation and inversion, and marsupializing the lower portion, thus making a skin fistula through which feedings could be given. Technically, it was an easy operation. By resecting the left fourth costal cartilage and a portion of the rib and dividing the intercostal muscle down to the pleura, it was easy to separate the parietal pleura from the mediastinum. By retracting the pleura and lung laterally, the mediastinum was easily entered. The trachea was identified by palpation, and the fistulous end of the esophagus was readily located by elevating the trachea, care being taken not to damage the vagus nerve. In this

way the tracheal stoma was closed, and the esophageal fistula could be used as an entrance into the stomach for feeding, and thus regurgitated matter would no longer enter the trachea.

The operative procedure was done entirely under local anesthesia. Even this premature baby withstood the operation well but succumbed in twelve hours, which was to be expected. I believe this is a feasible procedure, although in these infants with congenital malformations the mortality is essentially very high.

In connection with Dr. Lerche's paper, it explains a phenomenon that I have had to battle with in a friend and patient of mine. This patient was a perfectly well individual until she fell down a long flight of stairs and sustained a fractured skull. After the injury she had no esophageal symptoms for nine months. Following this period she had difficulty in swallowing, and under the fluoroscope a bolus of barium could be followed to the junction of the middle and upper third of the esophagus, where it temporarily lodged. This gave the patient considerable distress. The swallowing of a liquid would dislodge the bolus and it would proceed to the stomach in normal fashion. I could see no reason why it should lodge at this particular place until I heard the description of the anatomy of the esophagus by Dr. Lerche.

This patient was esophagoscoped on two occasions, and we were unable to find any ulceration or other objective findings. She has, however, not been able to swallow solid foods and has constantly to wash down the bolus with a swallow of liquid.

I should like to ask Dr. Lerche whether there is any difference in the innervation of the upper third of the esophagus as compared with that of the lower.

DR. OWEN H. WANGENSTEEN.—I want to comment on the presentation of Dr. Leven, which I look upon as being a real contribution to the therapy of the formidable disorder of stenosis of the esophagus complicated by the presence of a tracheobronchial communication. Most of us have shared the impression that this deformity is an absolutely hopeless entity, and certainly up to the present it has appeared to be such. I recall an occurrence relating to the first patient upon whom Dr. Leven did this operation. Some one said, "Why not let the poor infant die without having to tolerate the assaults of the surgeon?"

This simple expedient which Dr. Leven has described for us emphasizes the importance of practical contact with problems for the origination of ideas. One cannot sit down and abstractly think them out. We had frequently discussed together measures which might obviate the recurrence of regurgitation from the stomach into the esophagus and in turn into the lungs. In an earlier case operated upon by me, I had inserted a catheter into the lower esophagus through a gastrostomy opening at the time of operation, continuous suction being applied to the catheter. This agency, however, proved ineffectual in preventing regurgitation into the lungs.

With reference to the problem which still confronts Dr. Leven in dealing with this second case, it occurs to me that he would do well to close the present gastrostomy opening at an early convenient date, return the stomach to the peritoneal cavity, and establish gastrostomy for feeding through a small lateral stab wound.

There is another feature of the problem that is interesting. This concerns the likelihood of children and particularly infants, who have an indwelling catheter, acquiring an infection of the middle ear. In hundreds of instances in adults, an indwelling duodenal tube has been in the nose for days and occasionally for weeks. In no instance, as far as I know, has an otitis media developed, or has the patient developed pain in the ear. The discomfort felt in the throat incident to the presence of the tube is, of course, a matter of common experience. In infants, especially, as Dr. Leven has emphasized, the horizontal plane of a short eustachian tube favors infection.

Were it not for this occurrence, the indwelling duodenal tube might be put to more frequent use in infants. One of these I have previously described, and relates to its use in so-called congenital hypertrophic pyloric stenosis (Discussion, Transactions of Western Surgical Association, 1934, pp. 170-180). This disease is in fact badly named, for there is no actual stenosis, there being essentially only hypertrophy of the circular muscle of the pyloric sphincter. It is well known that infants who can be carried along on medical management until the fourteenth to sixteenth week of life frequently overcome the handicap of this anomaly spontaneously. It must, therefore, in fact be a growth disturbance, which maladjustment can, with the lapse of time, be overcome by the organism. With the cooperation of Dr. McQuarrie, professor of pediatrics at the University of Minnesota, several such infants who developed symptoms of pyloric obstruction for the first time between the sixth and tenth weeks of life were carried along satisfactorily by gastric feedings through an inlying urethral catheter in the nose. The frequent development of otitis media, however, necessitated discontinuance of the practice. It did not seem to make much difference whether the tube was led into the stomach through the nose or through the mouth. In no instance did the tube go through the pylorus, but the drop-by-drop feeding permitted the stomach to evacuate its content satisfactorily. It was quite evident from this experience that one reason the obstructed stomach regurgitates is that too much is put into it at one time.

DR. H. R. DECKER.—Dr. Truesdale has raised a question which is of considerable interest at this time, because there are some doubters in the field of pathology who say there is no such thing as heterotopic gastric mucosa. What is presumably heterotopic gastric mucosa, they say, is nothing more nor less than a normal glandular entity. It does not seem so, when the gastric juice will affect those glands selectively and cause ulceration in them. I cannot mention the name of the pathologist who reported this; but, if he is right in his concept that there is no heterotopic gastric mucosa, we shall have to drop that idea of it and think in terms of simple esophageal ulcers that are influenced by the gastric juice; and instead of peptic ulcers perhaps, we shall have to think in terms of these ulcers being secondary to acute esophagitis, rather than being definitely peptic.

Dr. Jackson and Dr. Chevalier L. Jackson have probably had the largest experience in the country with esophageal ulcer. When they reported a series of 88 ulcers, 27 active and 61 scars, or something like that, it represents considerable experience. Yet I recall that in this series there were only one or two that had any association with gastric and duodenal ulcer, and in looking through the literature I find there are not more than half a dozen cases reported with that association. In our case I jumped at the conclusion that we were dealing with a peptic ulcer of the esophagus.

Dr. Lilienthal raised the question of treatment by esophagogastrostomy. This is an admirable procedure, but it takes Dr. Lilienthal's trained hand. The average surgeon could not get along with it.

The situation described by Dr. Coryllos is apparently functional, and is not connected with organic disease of the esophagus. He referred to the three cases that Dr. Scrimger reported five years ago. My impression is that Dr. Scrimger decided, after losing one patient, since obviously there was a great chance of fistula, to drop that operation and do a modified procedure which simply sections the adjacent part of the diaphragm at the point where the esophagus goes through. The last case in which he carried out that procedure was a very distinct success.

STERILIZATION OF THE AIR IN THE OPERATING ROOM BY SPECIAL BACTERICIDAL RADIANT ENERGY

Results of Its Use in Extrapleural Thoracoplasties

Deryl Hart, M.D.
Durham, North Carolina

IN THE early days of the Duke Hospital we had an occasional severe infection, usually with the *Staphylococcus aureus*, which we thought came from poor skin sterilization. These occurred almost entirely in large operative wounds such as thoracoplasties, radical amputations of the breast, nephrectomies, arthroplasties, etc. A very thorough preparation of the skin was adopted but without an appreciable change in the number of infections. The entire operating room technic was then checked by cultures, but no positive growth of *Staphylococcus aureus* was obtained except from the floors, walls, and ceilings. These were repainted frequently and washed daily but with little effect.*

It was then found that *a Petri dish of sterile blood agar exposed for one hour to the air in any operating room, while it was being used, and cultured for twenty-four to forty-eight hours (Fig. 1) showed many colonies of Staphylococcus aureus, at times as high as 78.* The total number of colonies of all organisms (predominantly *Staphylococcus aureus,* hemolytic and nonhemolytic, and *Staphylococcus albus,* Fig. 2) was occasionally as high as 150. (Fig. 1, *A.*)

This work was done in attempting to determine the source of infections in our operating rooms and without a complete survey of the literature. Since becoming interested in this question the literature has been reviewed, and we are familiar with the work of Meleney, Dandy, Brewer, Goff, Whipple, Davis, Hunt, and others. We do not claim originality in the work which has been done and refer the reader to the literature and the articles cited in the bibliography. We feel strongly, however, *that pathogenic organisms in the air contaminated by human beings are responsible for most of our operating room infections. This point, while referred to, has not been sufficiently emphasized. When mentioned it has been placed secondary to skin contamination and is not*

From the Department of Surgery, Duke University School of Medicine.

Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

*We are indebted to all members of the Surgical Staff, operating room personnel and administration for their whole-hearted cooperation. We wish particularly to acknowledge the suggestions and help given by Dr. C. E. Gardner, Jr., of the Surgical Staff, and Doctors Smith and Martin and Miss Poston of the Bacteriological Staff.

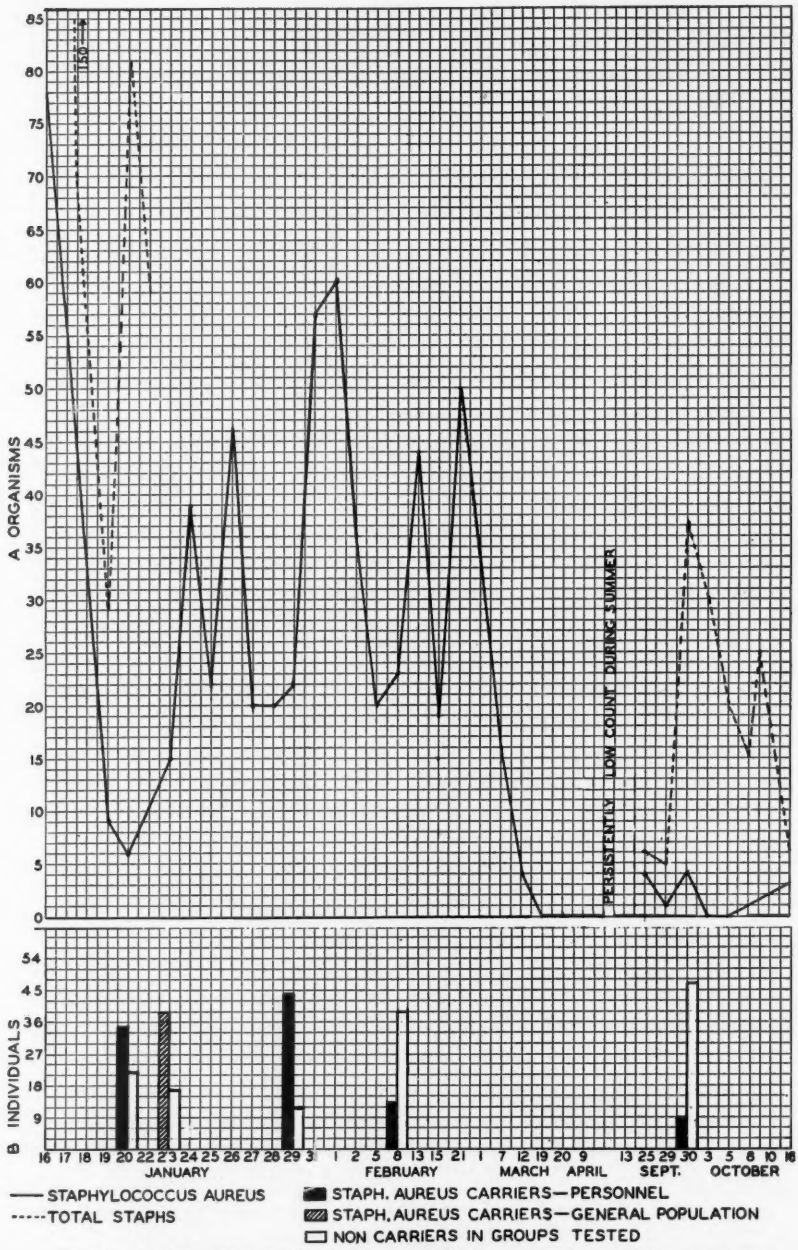


Fig. 1.—*A*, Air contamination in the operating room beginning before any steps were taken to reduce it and extending over the first nine months of this study. There was some reduction brought about by care in cleaning, isolating, and ventilating the rooms and masking all individuals entering, but the greatest drop came with the summer and fall months.

B, The number of carriers (in the nose and throat) among the operating room personnel and in the general population. With the decrease in the number of carriers there was a drop in the air contamination.

generally considered to be of major importance. A number of years ago in the hospital in which I worked an outstanding bacteriologist spent a great deal of time trying to find a flaw in the operating room technic to account for the occasional infection. In making his report he stated that we had an "air-tight technic," but he completely ignored the contaminated air which I am convinced was the source of the infections.

Repeated cultures in the operating rooms at the Duke Hospital showed that the incoming air was practically sterile (Fig. 3). Every effort was made to keep air from the remainder of the hospital from reaching the operating rooms. It was also determined that the degree of air con-

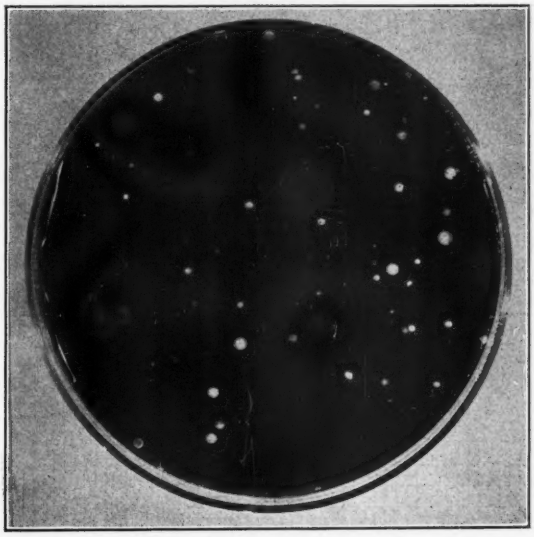


Fig. 2.—Petri dish of blood agar exposed to the air of an operating room for one hour during an operation and cultured for forty-eight hours. The colonies were identified as hemolytic *Staphylococcus aureus* 6, non-hemolytic *Staphylococcus aureus* 6, *Staphylococcus albus* 32. Before any steps were taken to reduce the air contamination, there were many more colonies on such a culture plate. Under such conditions the usual operation can be performed without great risk to the patient's life, but there is considerable risk of infection in operations of the magnitude of an extrapleural thoracoplasty.

tamination was much higher when the room was occupied than when unoccupied, even though in the latter case the air was kept agitated with electric fans. (Fig. 4.) Cultures of many locations throughout the hospital confirmed our impression that the degree of air contamination in any room was directly proportional to the duration of occupancy, the number of occupants, the percentage of carriers, and the intensity of the growth in their noses and throats (Fig. 1, *B* and Table I). After a much longer period of observation it was determined that there were great variations in the degree of air contamination and this contamina-

tion was more or less parallel with the rise and fall of the number of carriers in the operating room personnel and general population (Fig. 1). In general, the lowest air contamination was in the summer and

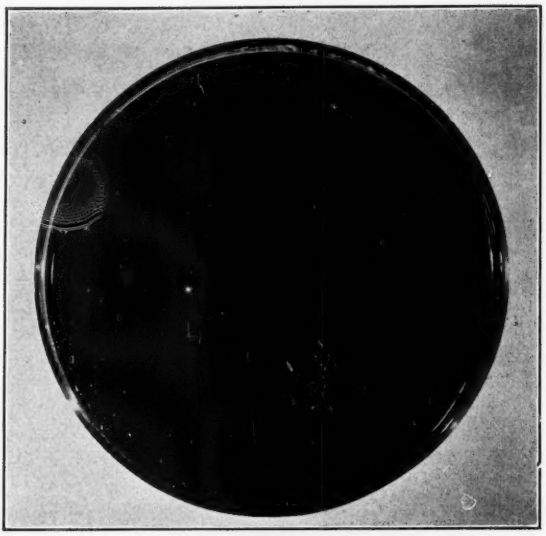


Fig. 3.—The washed air used for ventilating the operating rooms was sterile on most occasions when a culture was taken. This Petri dish of blood agar, exposed for one hour and cultured for forty-eight hours, showed only one colony of *Staphylococcus albus*. This may have reached the plate from the contaminated air in the operating room.

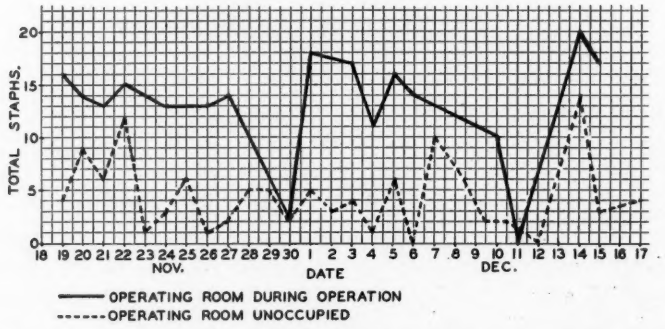


Fig. 4.—This graph shows that the air was much more heavily contaminated with staphylococci when the room was occupied than when unoccupied. When unoccupied the air was kept agitated with electric fans. Table I shows that in rooms less continuously occupied the air contamination was still lower.

fall months (Fig. 1), but there were wide variations during the winter and spring. (Figs. 5 and 6.) The peaks of contamination were associated with epidemics of respiratory infections.

The problem of masking is referred to in a number of articles, as those of Davis, Meleney, Walker, Whipple, Doust and Lyon, Kellogg and McMillen, Leete, Capps, Dannenberg, Mellinger, Weaver and others. These cite the inadequacy of the usual gauze masks, suggest covering the nose as well as the mouth, and describe new types of masks or a different mesh or thickness. None of the reports show that the air contamination with bacteria can be kept as low with masked human beings present as when they are absent. This, together with the comfort of the wearer, should be the criterion for a satisfactory mask from the point of view of preventing air contamination.

TABLE I

Cultures of the air by exposure of blood agar plates in various rooms throughout the hospital and medical school at a time when the bacterial contamination of the air in the operating rooom was high (see graph during January, Fig. 1, for cultures of the air in the operating rooms at the same time). The degree of contamination varied directly with the number of people present and the duration of occupancy. All the rooms were unoccupied when the cultures were made except the nose and throat examination room and office, the observation room, and the sterilizing room. These cultures together with those illustrated in Figs. 1 and 6 made us feel that the degree of air contamination was dependent on the duration of occupancy, the number of carriers, and the extent of the growth of organisms in their noses and throats.

DATE		LOCATION	DURATION OF EXPOSURE HOURS	STAPHYLOCOCCUS AUREUS	STAPHYLOCOCCUS ALBUS
January	19	Accident room	1	0	3
	19	Corridor Medical School	1	0	5
	19	Autopsy room	1	0	9
	19	Nose and throat office	1	8	18
	19	Nose and throat examination room	1	8	7
	20	Observation room, students present	1	12	60
	20	Sterilizing room	1	1	30
	20	Delivery room No. 1	1	0	2
	20	Delivery room No. 2	1	0	3
	20	Gynec. examination room	1	0	6
	21	Instrument room	1	0	0
	21	Ventilator duct	1	0	0

We attempted to eliminate all carriers from the operating rooms but found this to be impractical because of rapid changes in the nose and throat cultures. All persistent carriers with a high contamination were eliminated until free of staphylococci or streptococci. They were treated by vaccines and the nose and throat sprayed with bacteriophage.

An attempt was made to reduce the air contamination as much as possible by replacing the contaminated air with washed clean air. The bacterial count could be definitely reduced (Fig. 7), but when the room was occupied the air contamination rapidly rose. Only clean cases were allowed in the operating rooms used for large operations, all persons

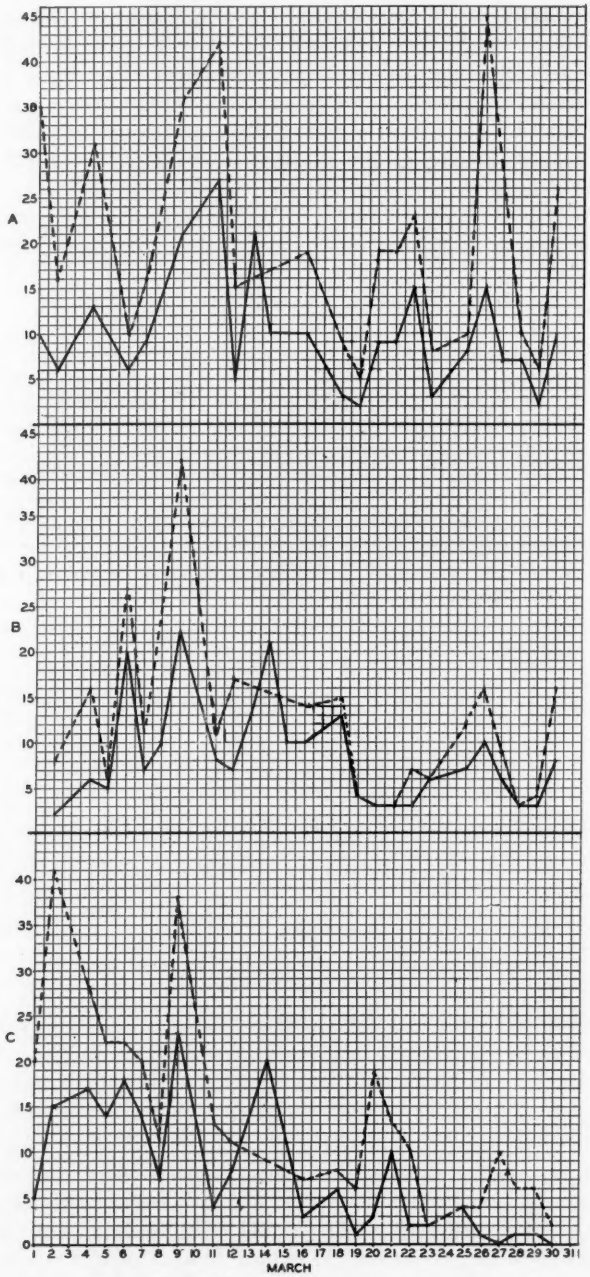


Fig. 5.—(See opposite page for legend.)

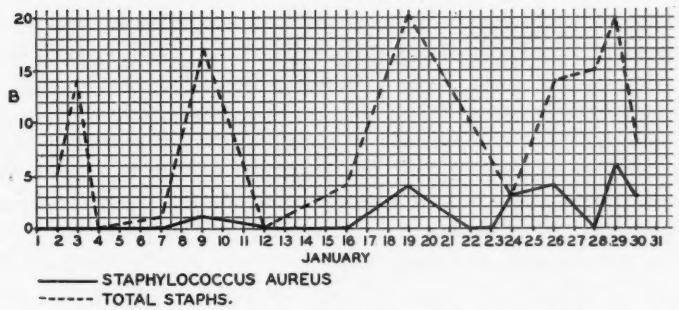


Fig. 6.—Periods of low air contamination occurred even in the winter months. The more persistent drop, however, occurred during the summer and the fall.

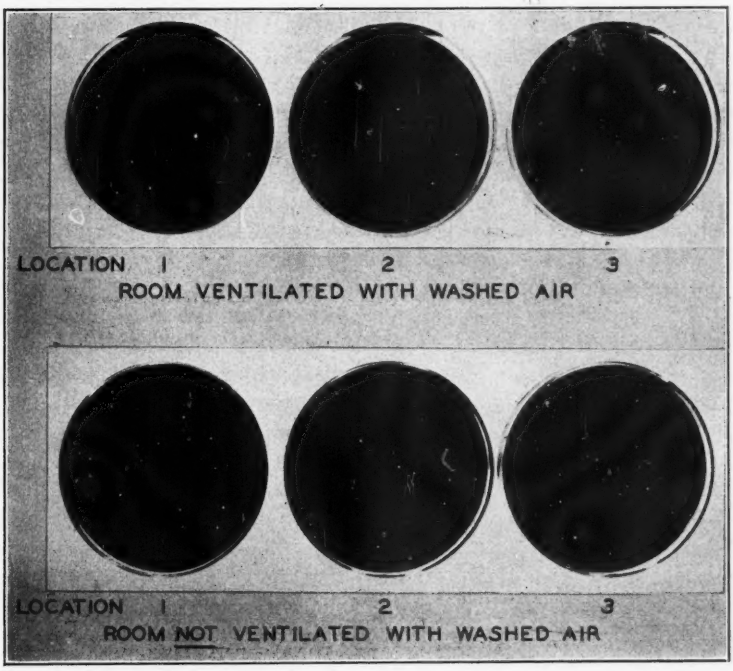


Fig. 7.—Twenty-four-hour cultures of sterile Petri dishes of blood agar exposed in the operating rooms for one hour with the room unoccupied except for placing the plates. Every precaution had been taken to keep the air as pure as possible. The lower plates were exposed first and then the ventilating fans were turned on and the upper set of plates exposed. This shows clearly the reduction in the number of organisms in the air by this washing out of the rooms with clean air. When people were present the reduction was not so great.

Fig. 5.—There were peaks of air contamination in spite of every precaution that we could take (compare with Fig. 6). These were most pronounced during epidemics of respiratory infections, and at such times the danger of infection in operative wounds was greatly increased. The peak of air contamination illustrated here is one of the highest we have had during the past three years, since we became air conscious. We have kept such graphs during this time in attempting to anticipate the periods of greatest danger for large operations. See Figs. 1, 4, and 6 for other sections of this graph. For evidence of the correlation of the air contamination with the nose and throat infection in the operating room personnel and general population see Fig. 1.

entering wore a mask over the nose and mouth at all times, and during all thoracoplasties double masks, each made of six thicknesses of 28 × 32 mesh butter gauze, were worn. By these measures we reduced the air contamination by from 60 per cent to 80 per cent, but this was not enough to prevent an occasional infection. Many will consider that the large number of staphylococci in the air is peculiar to our location. This is definitely not the case, since every report we have seen (Meleney in New York, Davis in Baltimore, Hunt in New England, etc.) gives a higher contamination of the air with these organisms than we have had during the past two to three years, during which period we have made a concerted effort to keep this contamination down.

We realize, of course, that other organisms, such as the streptococcus, are present at times in the noses and throats of large numbers of the population, and this organism gives rise to epidemics of operating room infections. The surgeon is much more likely to become upset by a streptococcal infection. The staphylococcus infection is more likely to be ignored, since it is usually considered to come from a contamination by the organisms on the skin. At most it may incite a change in the skin preparation or scrubbing technic, while the real source of the contamination (from the air) goes unsuspected. After our first gross infection with purulent drainage we extended the skin preparation to include ten minutes with soap and water, followed by benzine, alcohol and ether. The skin was then painted with two layers of 3½ per cent alcoholic solution of iodine, each allowed to dry and then thoroughly washed off with 70 per cent alcohol. Still having an occasional gross infection with the *Staphylococcus aureus,* we gave up local anesthesia. We felt that the deeper tissues might be inoculated by passing the needle through infected hair follicles and sweat glands in the skin. All wounds were carefully protected from the skin as soon as the skin incision was made. We then came to a realization of the fact that the air rather than the skin was the source of the infections. Any one who doubts the tremendous importance of this air contamination needs only to compare a culture plate exposed to the air for an hour with one inoculated from thoroughly cleaned skin to determine where the greater number of organisms are found.

Because of the contamination of the air, all extrapleural thoracoplasties were postponed until the summer or fall months and then were operated on only when the cultures of the air showed the contamination to be low. (Figs. 1, 4, 5, and 6.) Before closure all wounds were thoroughly irrigated with 2,000 to 4,000 c.c. of physiologic salt solution. Some were painted with a dilute solution of gentian violet with the hope that this would inhibit the growth of gram-positive organisms. Despite all these precautions and regulations, infections occurred with such frequency that at times we were greatly discouraged.

After two to three years of effort to eliminate air contamination of wounds and almost in desperation* we turned to radiations of such wave lengths as are known to kill organisms. We first tried a therapy ultraviolet lamp and proved that we could kill a sprayed culture of hemolytic *Staphylococcus aureus* at a distance of eight feet from the lamp within sixty seconds. The carbon arc lamp was tried but had practically no effect on the organisms.

In the fall of 1934 various manufacturers of ultraviolet lights of the character referred to above were asked to cooperate in the experiments to the extent of furnishing the necessary equipment. A favorable contact was made with the Westinghouse Lamp Company, which has conducted extensive researches in the generation and application of radiant energy, specializing in those regions of the spectrum which were found to be bactericidal. We are greatly indebted to them for their cooperation. They advised us as to the wave lengths and intensities of radiation which were most efficacious for killing bacteria and least irritating to the tissues, and supplied specially designed radiation tubes and other equipment for experimental purposes.

It is well recognized that the germicidal and fungicidal action of radiant energy depends upon the wave length of the radiation reaching the organisms. The destructive action of this radiation, such as the erythemal effect when living tissue is exposed, also is a function of the wave length and the amount of exposure.

It is at once evident that where long exposures are necessary, such as for lengthy operations, it is not only desirable but imperative that the nature of the radiations (wave length distribution and intensity) used for bactericidal action must be so selected that under all conditions a harmful burning action is avoided.

In designing a source of radiant energy which would prove effective in destroying bacteria, care had to be exercised to see that the intensities of the wave lengths capable of producing detrimental effects, such, for example, as erythema, were so low that such effects were practically nil. It was also of importance, however, to provide a sufficiently high level of bactericidal radiations to accomplish destruction of the bacteria without the intensity being of such a character as to create any discomfort to the patient. To meet these requirements a special radiant energy device was designed and constructed by the Westinghouse Lamp Company. These devices are tubular in shape, have unheated electrodes, and utilize a special gas mixture in which the discharge takes place with the production of radiant energy of the character mentioned above. During operation the tube remains at a temperature only a few degrees above room temperature.

*We were planning to try immunization of all patients for extrapleural thoracoplasty with the *Staphylococcus aureus* (hemolytic and nonhemolytic) if the bactericidal radiation had not been satisfactory.

An operating room was equipped with eight of these tubes, each 30 inches long, as shown in Fig. 8. Blood agar plates sprayed with hemolytic *Staphylococcus aureus* and a mixture of organisms were exposed to the radiation in the approximate position of the operative wound. These were about five feet from the individual tubes and four feet from the

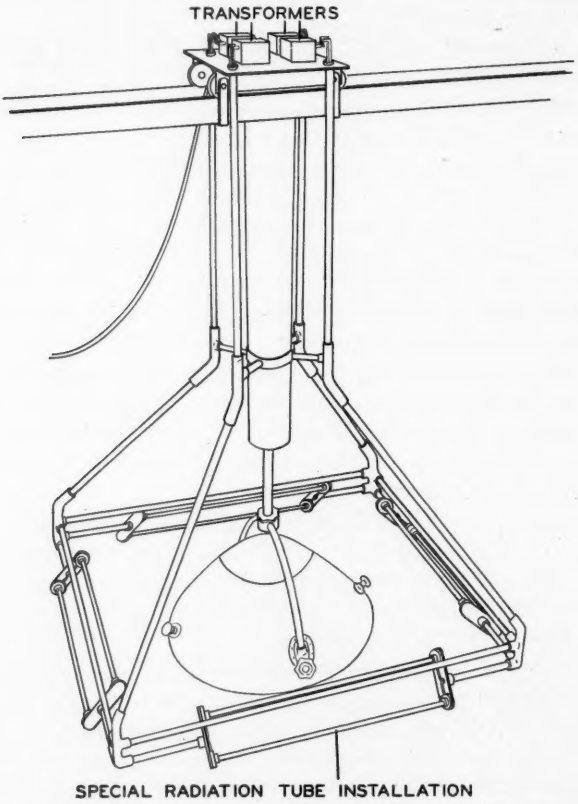


Fig. 8.—Arrangement of the tubes giving off bactericidal radiation as used in the following experiments and operations. Eight tubes, each 30 inches long, were grouped about the operating room spot light. The distance between the opposite tubes was five feet. The plane of the cluster was four feet above the operative incision or cultures, and this point was approximately five feet from the individual tubes. The supply and instrument tables were within the effective range of this radiation. This arrangement was made with the idea of sterilizing the air of the entire room if possible, but with the definite plan of laying down a barrage of bactericidal radiation of such an intensity that any organisms floating in the air would be killed before they could settle in a protected position on the supply and instrument tables or in the wound. All supply tables were stacked within the effective radius of this radiation and kept covered when not exposed to the radiation.

center of the cluster. The lightly sprayed cultures were killed within sixty seconds (Fig. 9) and the heavily sprayed cultures within less than five minutes (Fig. 10). Control experiments showed that the radiation

did not affect the culture media. Sprayed cultures were then exposed at distances up to 10 feet from the lights, with the results shown in Fig. 11.

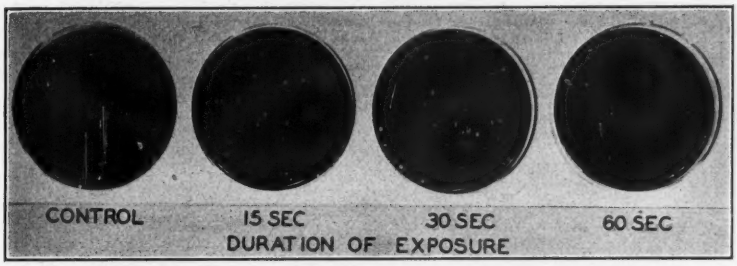


Fig. 9.—Forty-eight-hour culture of blood agar plates lightly sprayed with a filtered suspension of hemolytic *Staphylococcus aureus* and exposed to the eight tubes in the approximate position of an operative wound (five feet from the individual tubes). All organisms were killed within sixty seconds of exposure.

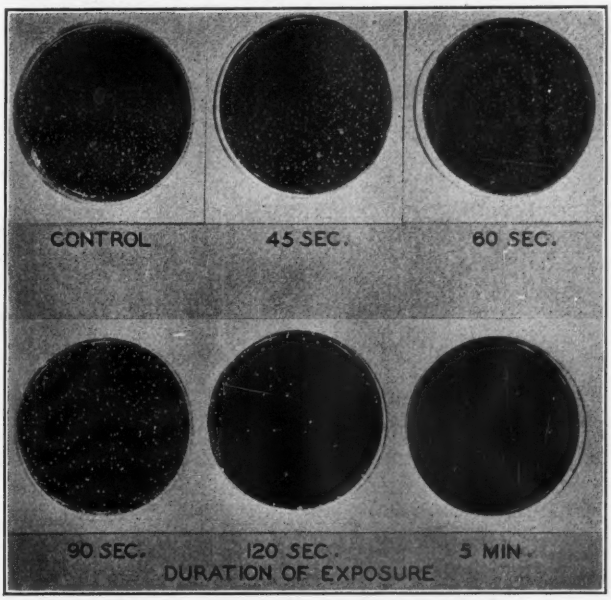
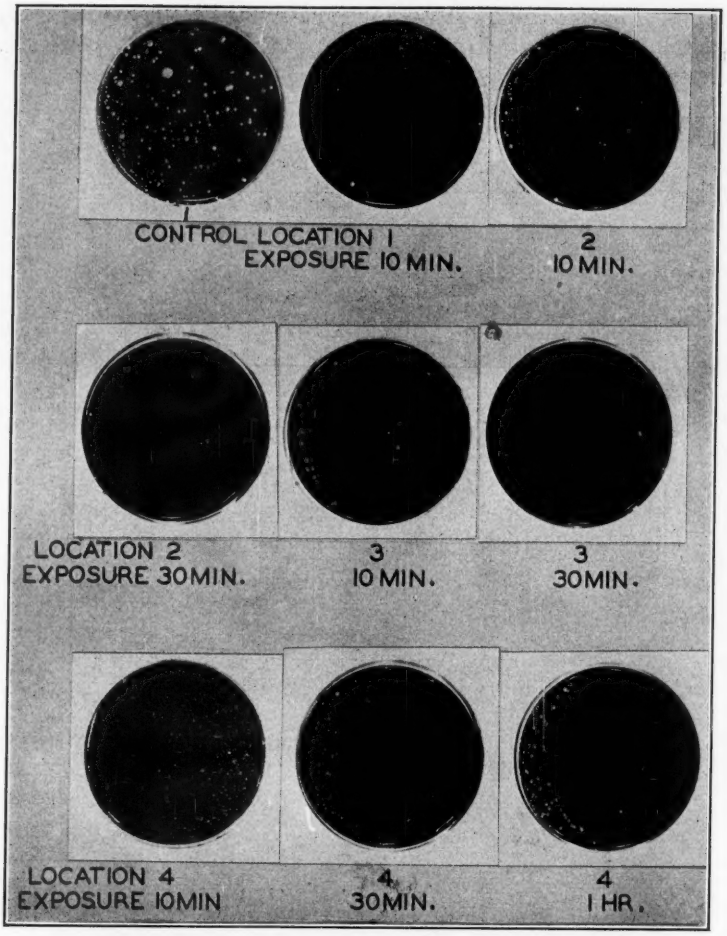


Fig. 10.—Heavily sprayed cultures of an unfiltered suspension of *Staphylococcus aureus* required over two minutes and less than five minutes for destruction of all but one colony of organisms. The experiment was similar to that illustrated in Fig. 9. It required longer to kill the organisms about the periphery of the dishes where they were protected from the radiation of some of the tubes. Where part of the organisms are in clumps, as in this experiment, a longer exposure is necessary for complete sterilization, since some of them may be protected from the radiation.

Sterile Petri dishes of blood agar were then exposed during two successive operations at a distance sufficiently great to cover the supply and instrument table. The first plates without the radiation showed many colonies on the forty-eight-hour cultures; while those exposed with the



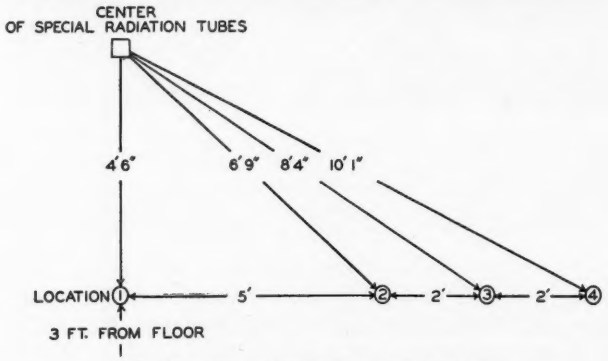
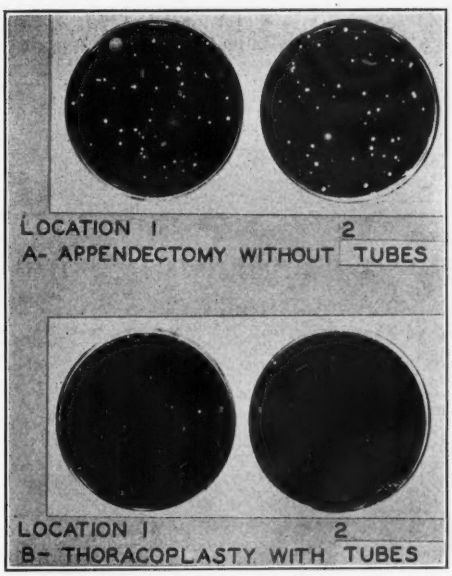


Fig. 11.—(See opposite page for legend.)



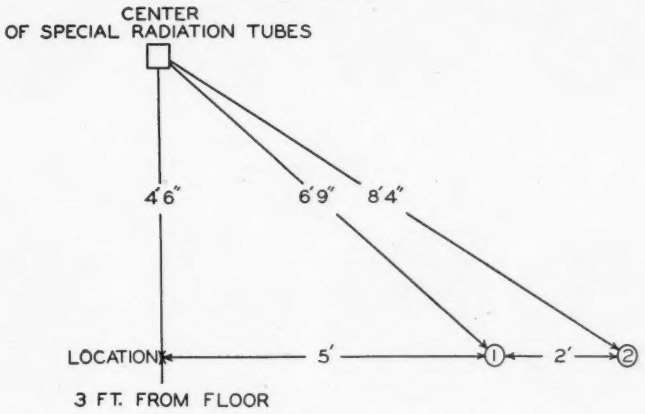


Fig. 12.—Forty-eight-hour cultures of Petri dishes of sterile blood agar exposed to the air of the operating room for one hour in the locations shown. Conditions were the same except for different patients and the difference in supplies and instruments.

A, Plates exposed the first hour during an appendectomy and without the radiation tubes.

B, Plates exposed the second hour with the radiation tubes when the contamination should have been greater. There were only three colonies on *B*, and one of these was near the rim where it may have been in a protected position.

Fig. 11.—Forty-eight-hour cultures of Petri dishes of blood agar, sprayed with a filtered suspension of hemolytic *Staphylococcus aureus* and exposed in the locations shown. The organisms nearest the tubes were quickly killed, while those at 10 feet were killed within less than thirty minutes. Some of those protected by the edge of the Petri dish were not killed within one hour.

radiation tubes on showed almost no growth (Fig. 12). Similar plates were then exposed in the extreme corners of the operating room 13 feet from the center of the cluster of radiation tubes. (Fig. 13.) These showed that at this distance, with the room occupied, the number of organisms falling out of the air could be greatly reduced, but not completely eliminated, within an hour of exposure to the radiation.

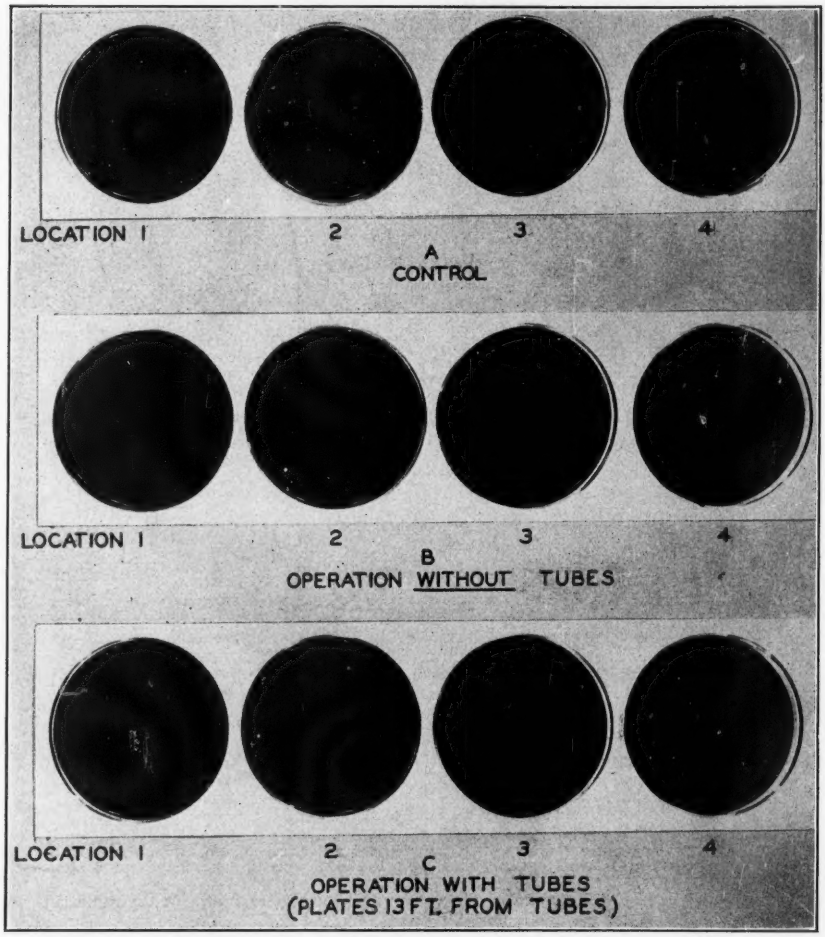


Fig. 13.—Twenty-four-hour cultures of sterile blood agar plates exposed in the corners of the operating room 13 feet from the center of the cluster of tubes (11 feet from the nearest tube). The three exposures were made under as nearly as possible similar conditions during three consecutive hours.

A, Without the radiation tubes burning while the room was being set up for the day.

B, Without the radiation tubes burning during the first operation.

C, With the radiation tubes burning during the second operation. At this time, with longer occupancy, the air contamination should have been greatest. The great reduction in the air contamination even at a distance of 13 feet during the operation with the tubes burning is clearly evident.

We felt, however, that we could lay down a barrage of radiation about the operative wound, supply and instrument tables of such an intensity that any organisms floating in the air would be killed before they could land in a protected position. We felt that if this could be accomplished without injury to the tissues, we could eliminate the occasional gross wound infection. By eliminating the wound contamination, which otherwise must always occur but which only rarely goes on to suppuration, we could make the postoperative course of all patients much smoother. Furthermore, there would be less temperature rise in those patients who had no infection before operation and possibly in other cases as well.

Wounds in rats were exposed to the radiation at five feet for thirty minutes and the abdominal wound and the peritoneum in dogs for

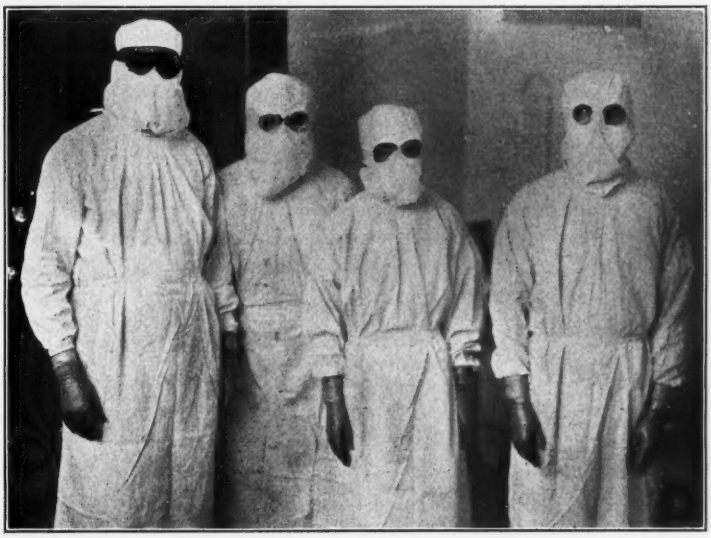


Fig. 14.—Photograph showing the protection used for the staff. The eyes were covered with goggles of plain glass which is impermeable. The hood made of closely woven cloth was heavily starched to fill the holes in the weave. An eye shade was also used by some, and more recently a sun helmet has met with considerable approval. The hood is hot, the goggles tend to become foggy and at all times limit side vision. The goggles can be kept clear and the hood can be made comfortable by placing a suction tube beneath the goggles to give better ventilation. Steps are now under way to provide a more comfortable protection for the operating room personnel.

ninety minutes without apparent damage. The wounds in fact seemed to heal better than where the radiation was not used. The peritoneal adhesions in dogs exposed to the radiation for ninety minutes were no greater than those in the control animals.*

Two blond volunteers were exposed to the radiation for eighty minutes and received only a slight reddening of the skin.

*Animal experiments performed with Dr. Paul Sanger, results to be published later. Many of the cultures were made by Dr. John Devine, who is working with me on the effect of the radiation on various pathogenic bacteria. To be published later.

It was imperative that the staff, who are frequently exposed for long periods of time, should be protected from the radiation. The eyes were covered with goggles of plain glass, which is impermeable, and the head with a hood of starched cloth as shown in Fig. 14. In addition some members of the team wore a sun helmet or eye shade. While this contrivance is hot and the goggles become fogged, it can be made quite comfortable by placing a suction tube beneath the goggles to insure adequate ventilation. The goggles of the type used limit side vision which is also some handicap. We are now working on a more comfortable type of protection for the staff.

From the first operation our results were most gratifying. The patients had less postoperative pain, the temperature was lower, and healing was more rapid. All wounds made in the field of this bactericidal radiation maintained throughout the period of healing a dry, scaly appearance.* (Fig. 15.) This was in contrast to the occasional moist, slightly macerated appearance of the suture line in certain thoracoplasties performed before the use of bactericidal radiation.

In the beginning only operations with large exposed areas were performed under the radiation tubes, but more recently they are being used for many of our cases. Only the extrapleural thoracoplasties performed in a clean operative field are considered here. Because of the magnitude of the operation, the size of the exposed area, the impossibility of meticulous hemostasis, the inevitable trauma, and the closure (in our hands) with catgut, they are among the group of surgical wounds that, when contaminated, are most favorable for the growth of organisms. A general review of all thoracoplasties is made here for the purpose of comparison. It should be noted that because of a severe infection in certain early cases which were not drained, we had made a practice of draining all wounds. This undoubtedly increased the possibility of the growth of organisms in the wounds and made it possible to obtain suitable material for culture. It also prolonged the time necessary for healing following operation. (Table IV.) In the first two cases performed with the bactericidal radiation, drains were inserted, but when withdrawn and cultured there was no growth. We then returned to our former practice of closing without drainage. The more rapid healing in recent cases (Fig. 15 and Table IV) is accounted for partly by the absence of the drainage tract. Of more importance, however, is the fact that there was no severe infection deep in the wound and not even enough contamination in the subcutaneous fat and about the sutures to give the moist, slightly macerated appearance with cutting of the sutures sometimes seen in previous cases.

Of 110 consecutive extrapleural thoracoplasties, usually with the removal of four to five ribs, performed on 61 patients, there were six

*In one case cultures of a suture removed ten days after operation showed pure growth of *Staphylococcus albus*, but there was primary healing of the incision.

deaths. One patient died of respiratory failure on the operating table during the second stage, after two ribs had been removed. He had stopped breathing during the induction stage of the anesthesia but had responded satisfactorily to artificial respiration. His condition was

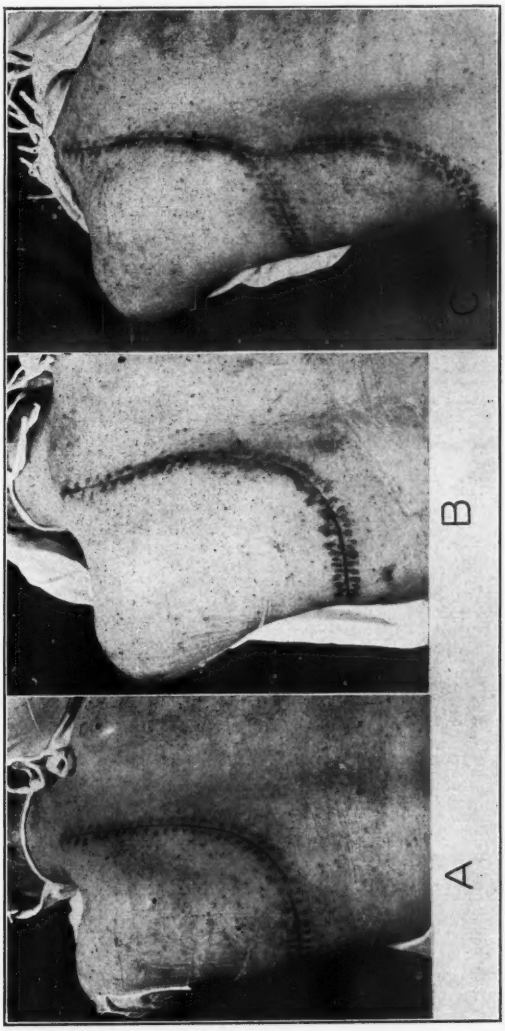


Fig. 15.—Photographs illustrating the scars of three successive operations for the removal of parts of eleven ribs (Fig. 20). The first rib in all cases was removed entirely, taking the transverse process and dividing the rib anteriorly at the costochondral junction. Most of the second and third ribs and the upper transverse processes were removed in all cases. The patient illustrated here was considered to be a poor risk and as a result the time between operations was longer than usual.

A, Picture taken eighteen days after the first operation, at which time three ribs were removed.

B, The incision for the second operation, performed nineteen days after the first, was through the scar of the first. This picture was taken one day after the sutures were removed and nine days after operation.

C, The scar for the third stage at its upper end extends into the scar for the two preceding operations. This picture was taken the day the sutures were removed, seven days after operation. The third operation was performed fifteen days after the second. We are afraid to open, at such an early date, wounds not performed under this bactericidal radiation; since there may be colonies of organisms persisting from the first operation, and these may be spread throughout the new incision.

thought to be satisfactory, and the operation had been continued. Another died seven days after operation with an extensive bronchopneumonia (autopsy showed gram-positive cocci) of the entire good lung. The other four patients all had an extensive wound infection with the

hemolytic *Staphylococcus aureus.* Two of these had a blood stream infection with the same organisms and died as a direct result of the infection. (Fig. 16.) Of the remaining two, one died five weeks after opera-

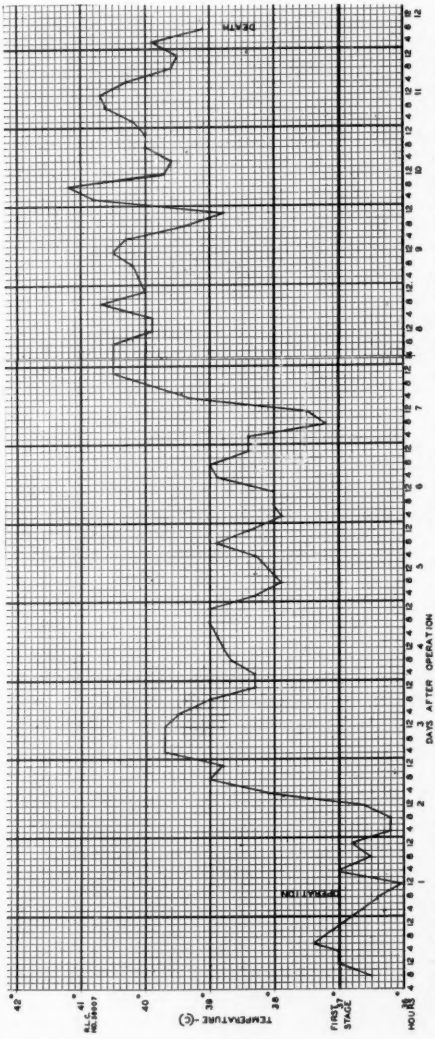


Fig. 16.—Temperature curve of a patient having a thoracoplasty wound (last thoracoplasty performed without bactericidal radiation) infected with hemolytic *Staphylococcus aureus.* The rapid rise of temperature the day after operation suggested infection in the wound. Cultures were made and the organism was identified. It was identical with the one which had been cultured from the air during the operation. The wound was opened widely and was found to be infected throughout. Even though free drainage was instituted the patient remained critically ill. The organism was recovered from the blood in large numbers, and the patient went rapidly down hill and died eleven days after operation.

tion of multiple pulmonary emboli, and the other died eight days after operation of bronchopneumonia involving the good lung and a pleural effusion on the operative side. In these two cases the infection was quite likely the direct cause of death.

The deaths, however, do not tell the entire story, since in many of the patients who recovered from the operation the convalescence was unduly

TABLE II

TEMPERATURE

Temperature elevations of patients operated on with and without the bactericidal radiation tubes for sterilizing the air. The greater number of patients operated on with the radiation tubes had a temperature below 38° C., while the majority of those operated on without this radiation had a temperature elevation of more than 38° C. Since we are attempting to give an idea of the general temperature curve rather than single peaks, we have not classed a temperature curve in the higher group unless there were more than two recorded temperatures in this division. For example, if there were only two recorded temperatures above 38° C. it would be classed in the 37° to 38° C. group, while if there were as many as three recorded temperatures above 38° C. it would be classed in the 37° to 39° C. group. This applies to the preoperative and postoperative temperatures with and without the radiation tubes.

BEFORE OPERATION					AFTER OPERATION			
PERCENTAGE OF OPERATIONS					PERCENTAGE OF OPERATIONS			
WITH RADIATION		WITHOUT RADIATION		TEMPERATURE GROUPS	WITHOUT RADIATION		WITH RADIATION	
%	NO.	%	NO.		%	NO.	%	NO.
100	(18)	88.2	(97)	37°-37.5° C.	2	(2)	44	(8)
0		10.8	(12)	37°-38.0° C.	30	(33)	28	(5)
0		1.0	(1)	37°-39.0° C.	53	(58)	28	(5)
0		0		37°-40.0° C.	14	(16)	0	
0		0		37°-41.0° C.	1	(1)	0	
100	(18)	100.0	(110)		100	(110)	100	(18)

Number of operations in parentheses.

TABLE III

DURATION OF TEMPERATURE AFTER OPERATION

The postoperative temperature curves are analyzed in regard to the time after operation when the temperature remained at or below the preoperative level. The greater number of the cases with the radiation tubes were normal within three days or less, while almost all those patients operated on without the radiation tubes had a temperature elevation for four days or longer.

	PERCENTAGE OF OPERATIONS			
	WITHOUT RADIATION		WITH RADIATION	
	%	NO.	%	NO.
0 Days (not more than two elevations above 37.5° C.)	2.0	(2)	44.5	(8)
2 Days	1.0	(1)	5.5	(1)
3 Days	10.0	(11)	16.7	(3)
4 Days	9.0	(10)	22.2	(4)
5-7 Days	32.0	(35)	11.1	(2)
1-2 Weeks	35.0	(39)	0	
2-3 Weeks	5.5	(6)	0	
3 Weeks +	5.5	(6)	0	
	100.0	(110)	100.0	(18)

Number of operations in parentheses.

stormy. Of the 61 patients, 52 were improved, 3 unimproved because of an extension of the tuberculosis, and 6 died. From 110 operative incisions a positive growth of organisms was obtained during convales-

cence from 36. (Hemolytic *Staphylococcus aureus* 28, *Staphylococcus aureus* 5, *Staphylococcus albus* 3, hemolytic streptococcus 1, *B. subtilis* 1, *B. coli* 2.) A staphylococcus was obtained from all but one of the infected cases, and in all severe infections the hemolytic *Staphylococcus aureus* was present.

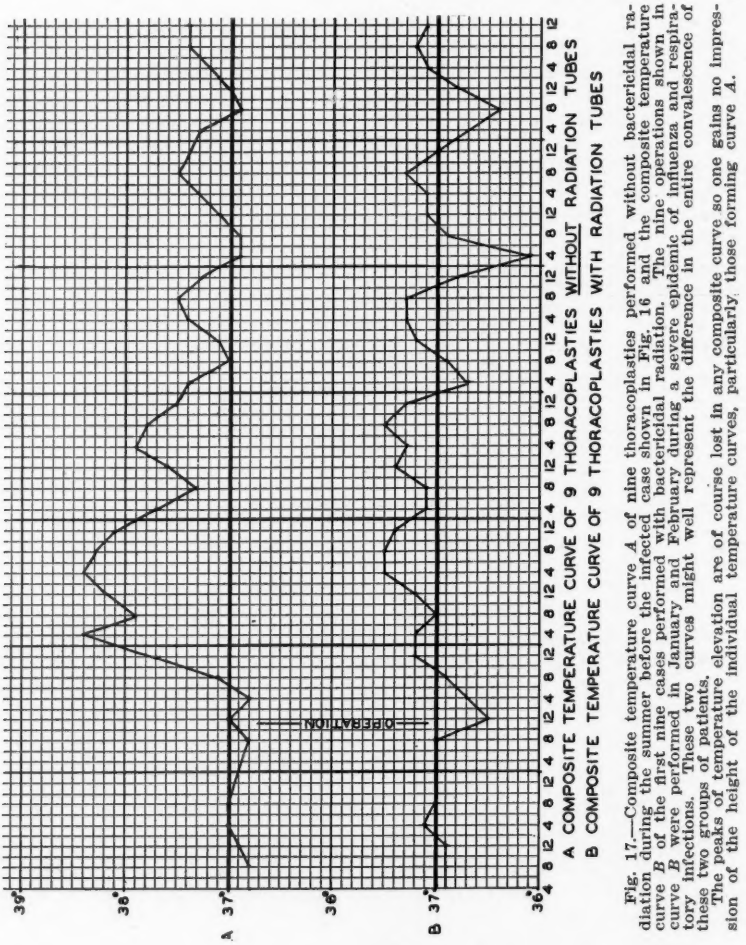

A COMPOSITE TEMPERATURE CURVE OF 9 THORACOPLASTIES WITHOUT RADIATION TUBES
B COMPOSITE TEMPERATURE CURVE OF 9 THORACOPLASTIES WITH RADIATION TUBES
OPERATION
39°
38°
37°
36°
A
B
4 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12 2 4 6 8 12

Fig. 17.—Composite temperature curve *A* of nine thoracoplasties performed without bactericidal radiation during the summer before the infected case shown in Fig. 16 and the composite temperature curve *B* of the first nine cases performed with bactericidal radiation. The nine operations shown in curve *B* were performed in January and February during a severe epidemic of influenza and respiratory infections. These two curves might well represent the difference in the entire convalescence of these two groups of patients.
The peaks of temperature elevation are of course lost in any composite curve so one gains no impression of the height of the individual temperature curves, particularly those forming curve *A*.

In looking back over the records it is our impression now that many of the cases recorded as uninfected would have shown a growth of organisms had a culture been taken. The temperature elevation of the patients showing a positive growth was frequently no higher and of no longer duration than for some of those in which an infection was not suspected and no culture was taken. Many of these cases with no cul-

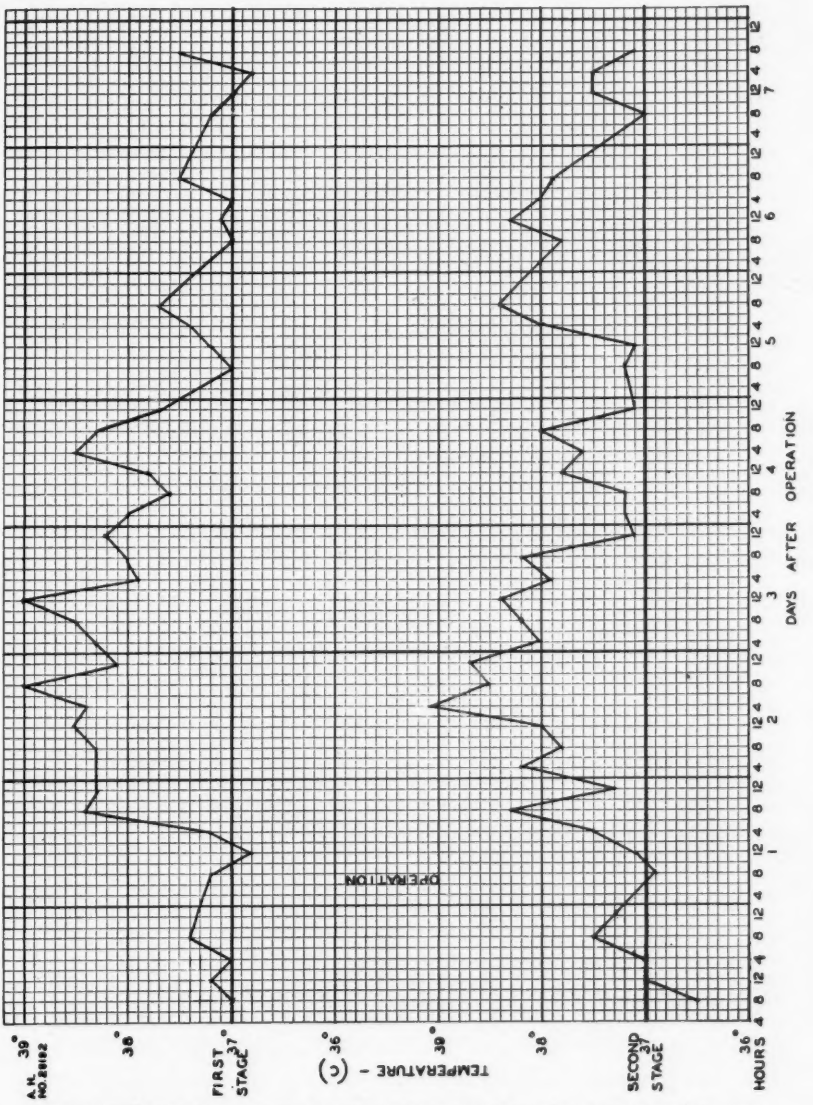


Fig. 18.—Temperature curve of an extrapleural thoracoplasty performed in two stages without bactericidal radiation. No cultures were taken from either wound, since they were not considered infected and they are classed in the group of non-infected cases. Both curves fall in the group of 37° to 39° C., elevations in Table II and the group of five to seven days' duration in Table III. Following the first operation, with resection of three ribs, the incision healed within eleven days and following the second operation, with removal of four ribs, within twenty-eight days.

ture and recorded as not infected showed a much higher and longer temperature elevation than is seen in the cases operated on under the radiation tubes.

The temperature curves of all patients are analyzed as to height reached after operation (Table II) and the duration before it was down to the preoperative level (Table III). In Table II it is readily seen that the temperature following 72 per cent of the 18 operations performed under the radiation tubes was 38° C. or less; while for the 110 operations performed without this radiation the postoperative temperature was above 38° C. in 68 per cent of cases. The differences in the duration of the postoperative temperature elevations as shown in Table

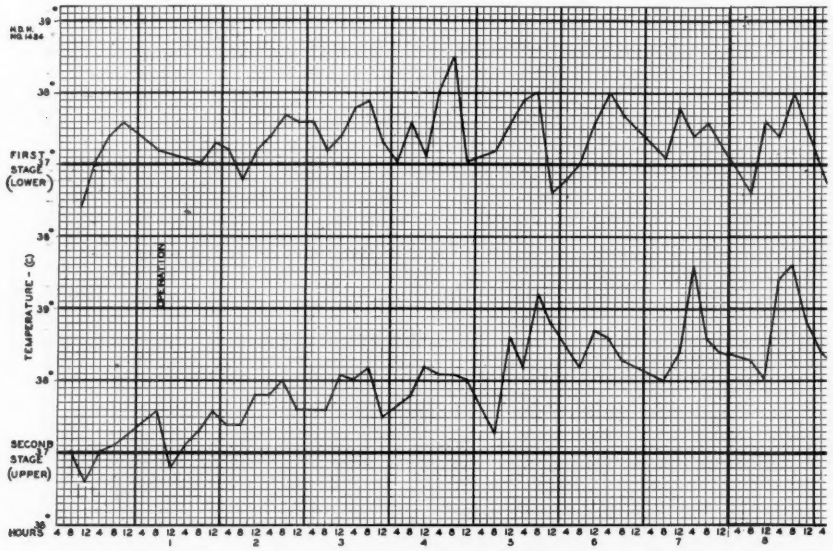


Fig. 19.—Temperature curves of an extrapleural thoracoplasty performed in two stages without bactericidal radiation. After the first stage the temperature, while not high, was elevated for twenty-one days. This temperature curve falls in the group 37° and 38° C., Table II, and the group of two to three weeks' duration, Table III. Following the second stage the temperature was high (group 37° to 40° C., Table II) and the wound was found to be extensively infected and was opened for free drainage. Fifteen days after operation the temperature was down to normal, and further recovery was uneventful. It was seventy-three days after operation, however, before the wound had completely healed.

III are even more striking. In 66.7 per cent of those operations performed under the radiation tubes the temperature was down to normal within three days or less; while for 87 per cent of the operations performed without the bactericidal radiation the postoperative temperature elevation was present for four days or longer. This great change in the temperature elevation and duration can be accounted for only by the elimination, or great reduction, of bacterial contamination of the wound.

In order to give a more accurate idea of the temperature elevation and duration following operation, certain representative cases are taken at random from the large group operated on without the radiation. The first (Fig. 16) shows the last patient operated on without the radiation. The temperature rose rapidly the day after operation and remained elevated until the patient's death ten days later. With this death we discontinued performing extrapleural thoracoplasties until after the installation of the bactericidal radiation tubes. In Fig. 17 there are two composite temperature curves: *A*, nine thoracoplasties performed during the summer immediately before the case illustrated in Fig. 16; and *B*, the first nine thoracoplasties performed after beginning the use

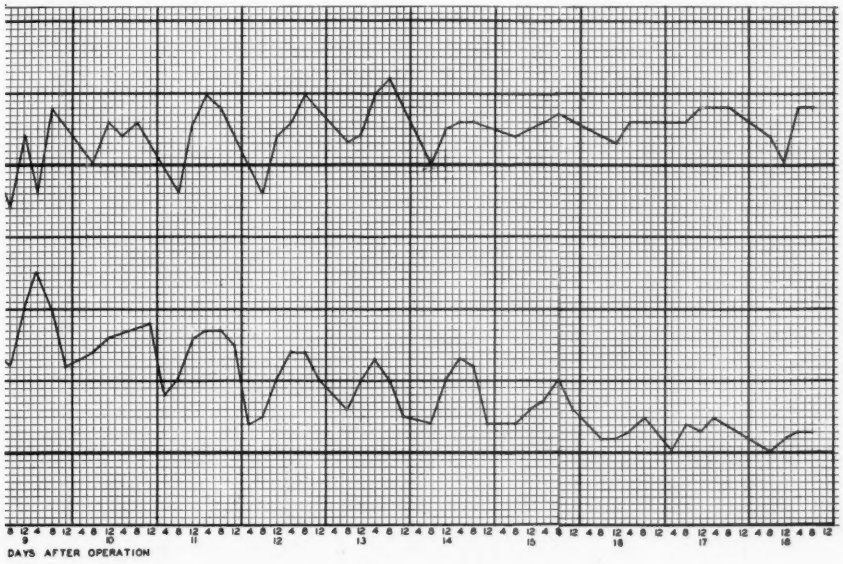


Fig. 19 Continued.

of bactericidal radiation. The latter operations were all performed in January and February during a severe epidemic of influenza. The wide excursions of temperature, particularly in the group without the radiation tubes, are necessarily lost in these composite curves.

The temperature curves for both the first and the second stages illustrated in Fig. 18 fall in the 37° to 39° C. group for temperature elevations, Table II, and the five to seven day group for duration of temperature, Table III. From our records this case must be classed as uninfected for both operations. We feel certain, however, that this degree of temperature, particularly for the second stage, was caused by bacterial contamination of the wound. Fig. 19 illustrates the long

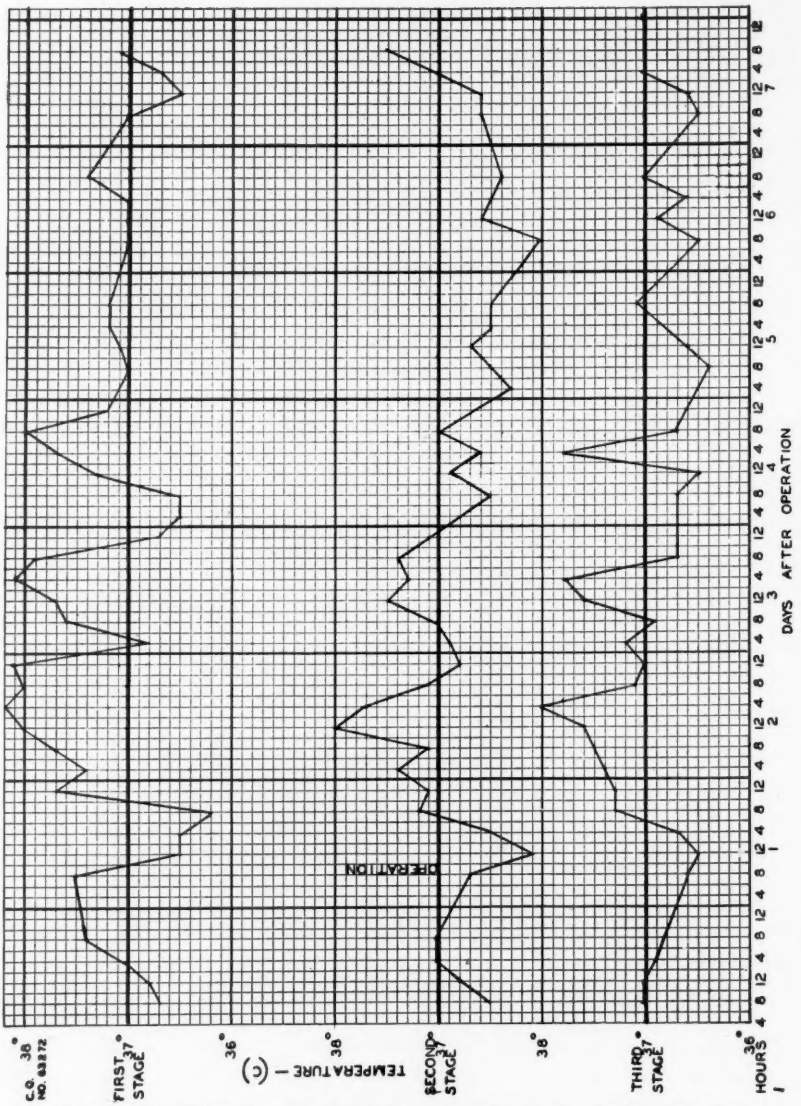


Fig. 20.—Temperature curves following an extrapleural thoracoplasty performed in three stages (photograph shown in Fig. 15) with bactericidal radiation. The patient was considered to be a poor risk.

Following the upper stage, with removal of three ribs, the temperature went above 38° C. on three occasions and was classed in the group 37° to 39° C., Table II, and was placed in the four day group, Table III. The sutures were not removed until the tenth day, and at that time the wound had healed.

The second stage was performed twenty days after the first, with the removal of four ribs. The temperature went above 37.5° C. on only two occasions, so the curve falls in the group 37° to 37.5° C., Table II, and the group of three days postoperative temperature elevations, Table III. The sutures were removed on the eighth day, and the wound was healed.

The third stage was performed fifteen days after the second with the removal of four ribs. The temperature was above 37.5° C. on three occasions, so it was placed in the group 37° to 38° C., Table II, and in the four day group for duration, Table III. The sutures were removed on the eighth day, and the incision was healed except for a little moisture around a suture from which a pure culture of *Staphylococcus albus* was obtained.

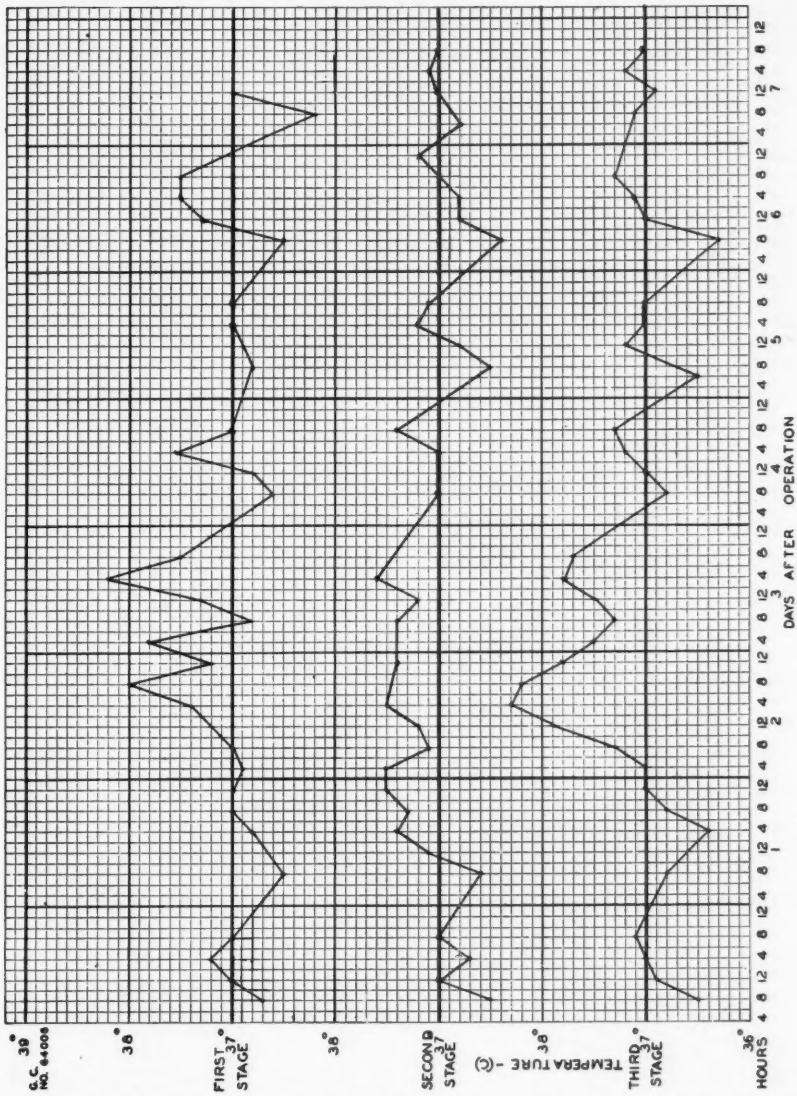


Fig. 21.—Temperature curves of an extrapleural thoracoplasty performed in three stages with bactericidal radiation.

Following the upper stage, with removal of four ribs, the temperature went above 37.5° C. on three occasions and above 38° C. on one occasion, so it falls in the group 37° to 38° C., Table II. The sutures were removed on the eighth day, and the wound was healed.

The second stage, with removal of four ribs, was performed nineteen days after the first. The temperature went above 37.5° C. on only two occasions.

The third stage was performed sixteen days after the second with removal of three ribs. The temperature went to 38.3° C. the day after operation, at which time the patient complained of a sore throat. This curve falls in the 37° to 38° C. group, Table II. The sutures were removed on the seventh day, and the wound was healed.

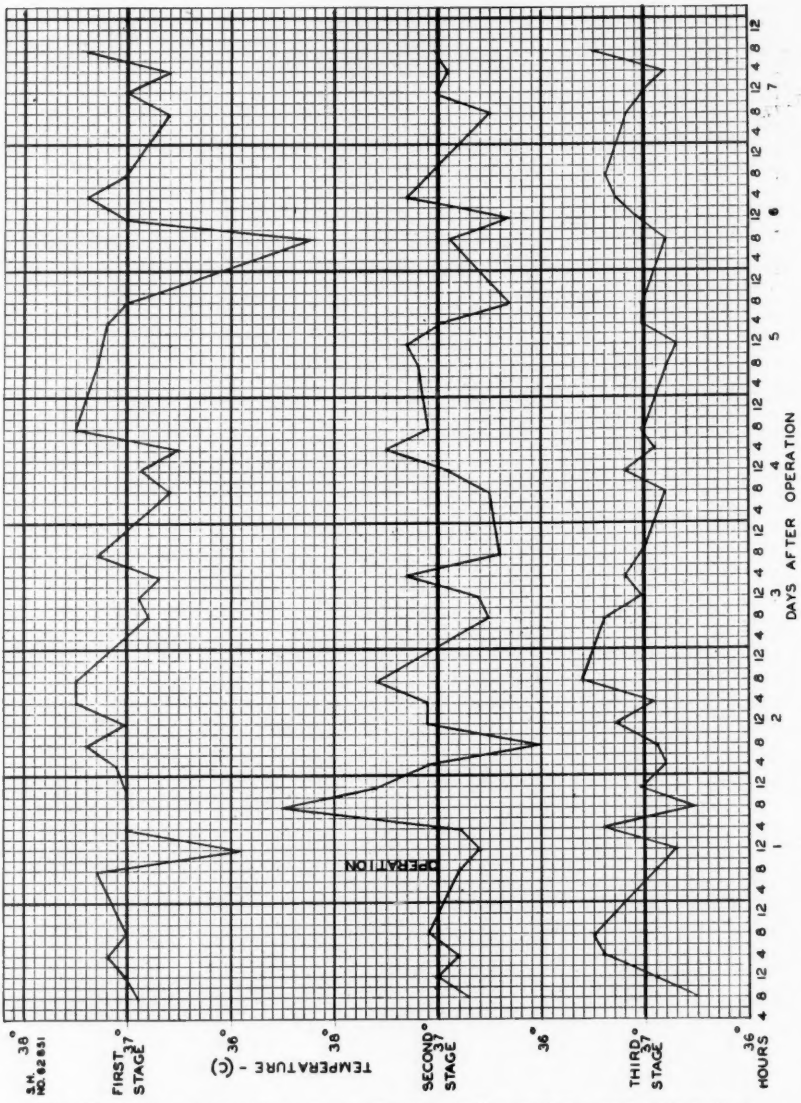


Fig. 22.—Temperature curves of an extrapleural thoracoplasty performed in three stages with bactericidal radiation. The patient had a persistent tachycardia before operation and was considered to be a poor risk.

After the first stage, with removal of parts of four ribs, the temperature did not go above 37.5° C.; the sutures were removed on the eighth day, and the wound was healed.

The second stage was performed nineteen days after the first with removal of parts of four ribs. Following this the temperature went to 38.5° C. following a transfusion the day of operation, and went to 37.6° C. on two other occasions, so this temperature curve was placed in the group 37° to 38° C. in Table II. The sutures were removed on the seventh day, and the wound at that time was healed.

The third stage was performed twenty-one days after the second with removal of parts of three ribs. The sutures were removed on the seventh day, at which time the incision was healed.

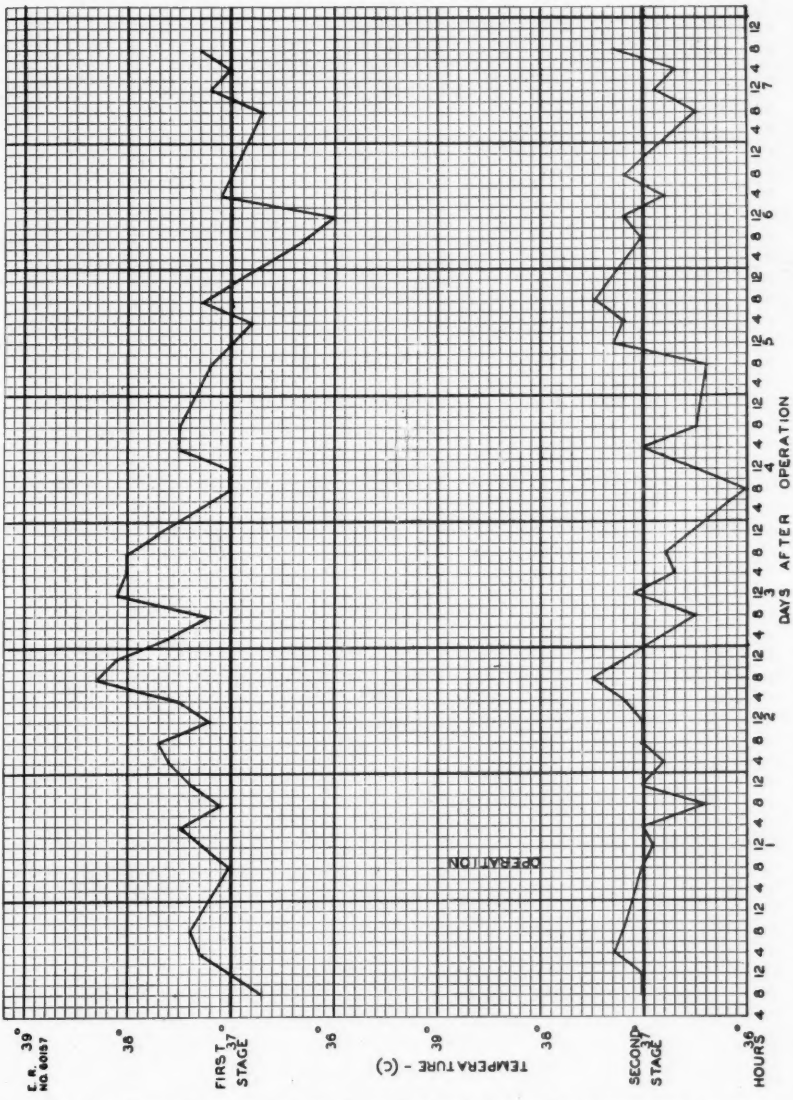


Fig. 23.—Temperature curves of an extrapleural thoracoplasty performed in two stages in a field of bactericidal radiation. At the first stage parts of the five upper ribs were removed, and at the second stage, three weeks later, parts of the next six.

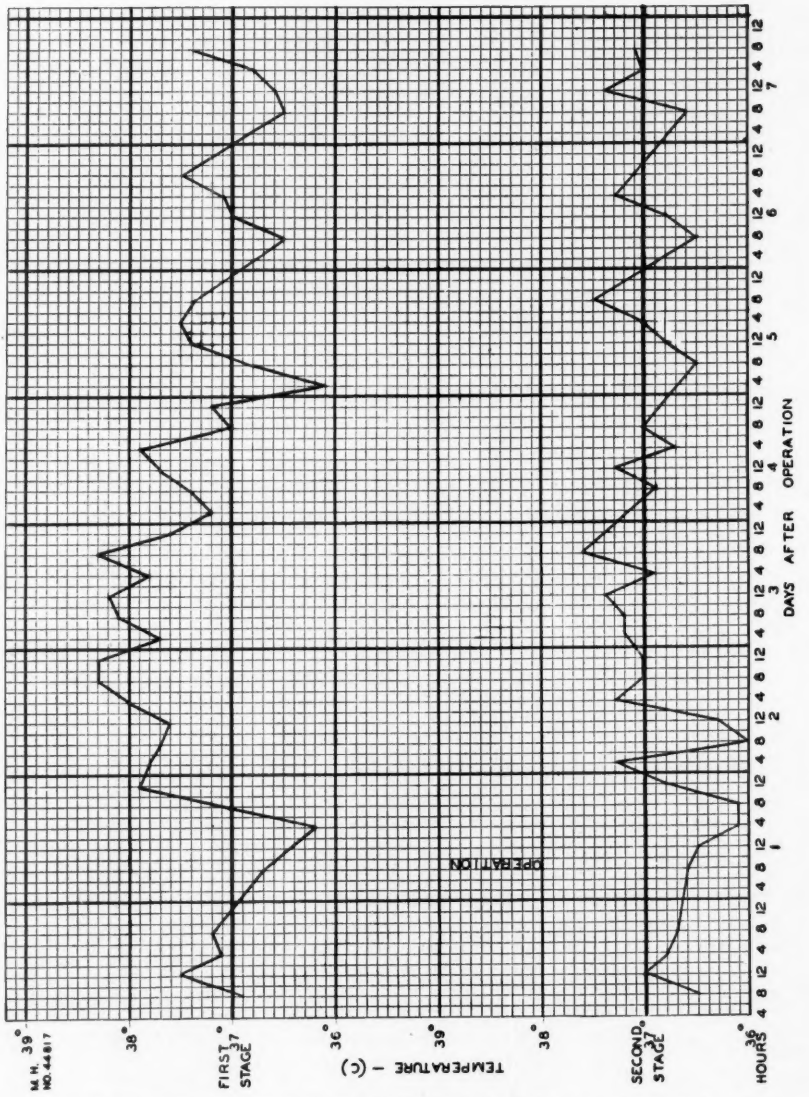


Fig. 24.—Temperature curve of an extrapleural thoracoplasty performed in two stages in a field of bactericidal radiation. At the first stage parts of the four upper ribs were removed and at the second stage parts of the next three.

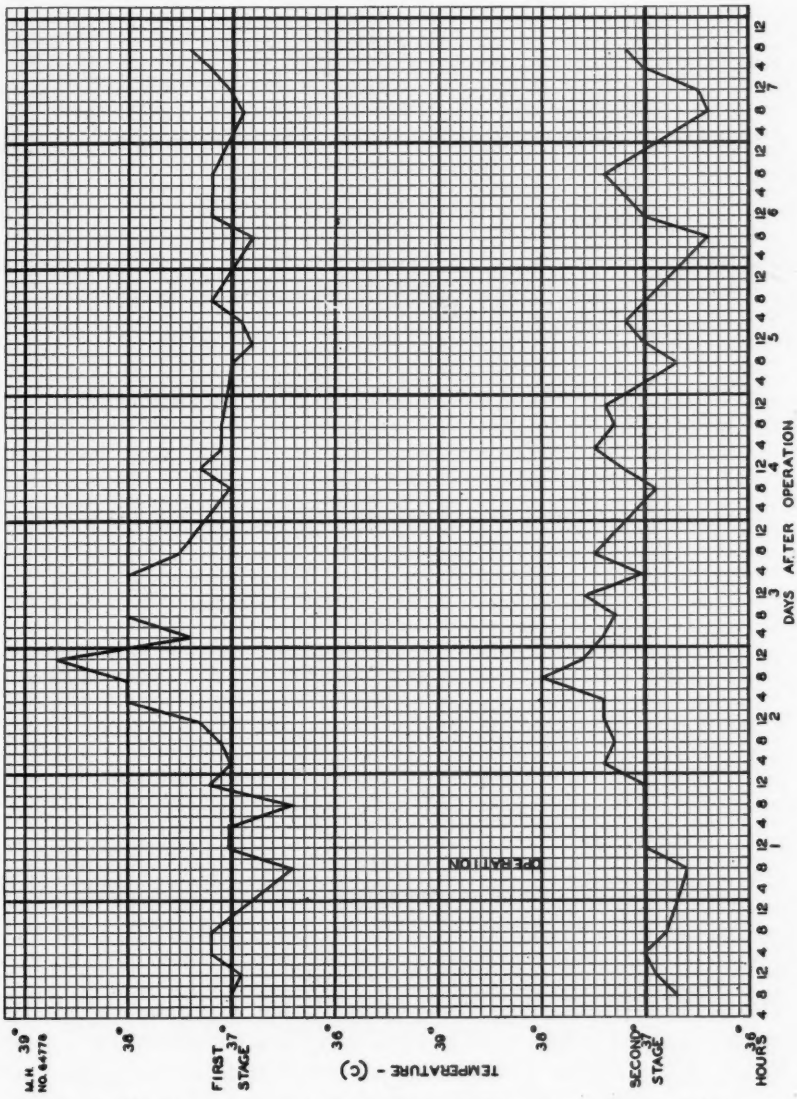


Fig. 25.—Temperature curve of an extrapleural thoracoplasty performed in two stages in a field of bactericidal radiation. At the first stage parts of four upper ribs were removed, and at the second stage, two weeks later, parts of the next four were removed.

duration of temperature following certain thoracoplasties performed *without bactericidal radiation* either with or without gross evidence of infection in the wound.

The temperature curves of all patients operated on in a field of bactericidal radiation are given for one day before and one week after operation, in order to show the elevations for the different stages of the collapse. (Figs. 20 to 28.)

The time required for healing is given in Table IV. The much shorter duration in the group performed with the bactericidal radiation should be interpreted in view of the fact that most of the wounds without the radiation were drained, while only 2 of the 18 with the radiation were drained. The drains were inserted, however, because of fear of infec-

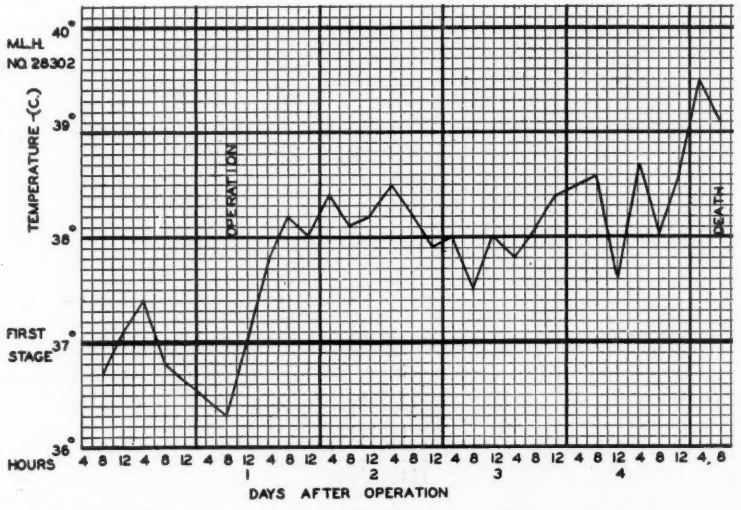


Fig. 26.—Temperature curve of an upper stage extrapleural thoracoplasty performed in a field of bactericidal radiation with removal of five ribs. The patient had a tuberculous laryngitis and twenty-four hours after operation developed marked respiratory distress which was relieved following the administration of carbon dioxide by coughing up a large plug of mucopurulent material. She had a recurrence of symptoms with progressively greater difficulty as a result of respiratory obstruction of this type. A tracheotomy was performed the night before death and the trachea kept aspirated frequently with transient relief. The wound was not infected.

tion. In many cases the prolonged period of healing was caused by the contamination of the wound. (Table IV.) In certain cases this caused only a profuse serous drainage immediately following operation, in others a purulent drainage over a longer period of time, while in some it was necessary to open up the wound widely and allow it to close by granulations.

The only death among the patients operated on in the field of bactericidal radiation was from asphyxia in a patient who had a tuberculous

laryngitis. This patient had a tracheotomy with temporary relief, but she also had asthmatic-like attacks, as is sometimes seen in patients with

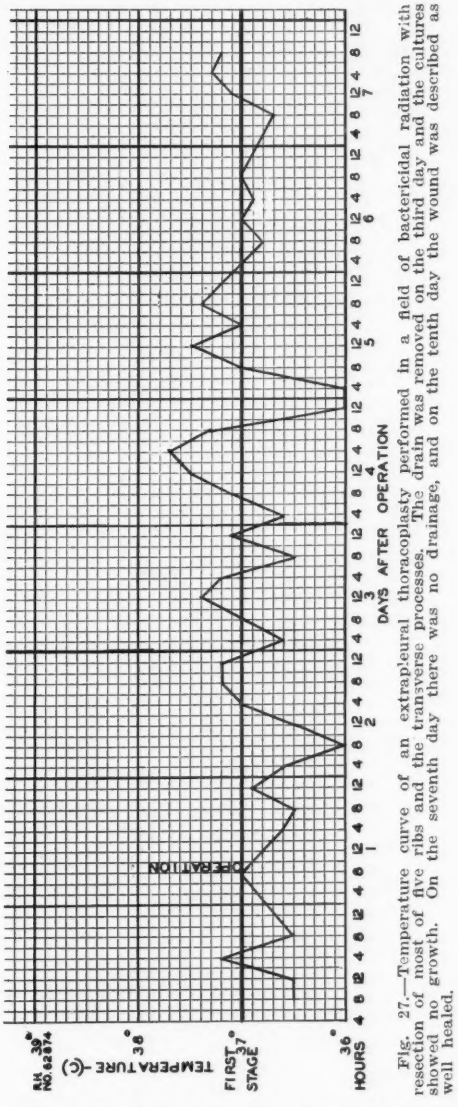


Fig. 27.—Temperature curve of an extrapleural thoracoplasty performed in a field of bactericidal radiation with resection of most of five ribs and the transverse processes. The drain was removed on the third day and the cultures showed no growth. On the seventh day there was no drainage, and on the tenth day the wound was described as well healed.

extensive tuberculous infection of the bronchi, and she died as a result of obstruction. A postmortem examination could not be obtained.

The improvement in results is best illustrated by the absence of infection in the 18 wounds made under the radiation, the much lower tem-

perature following operation, the earlier healing of the wounds, and the rapid return of the patients' general condition to normal. This is all the more striking when one considers that these operations were per-

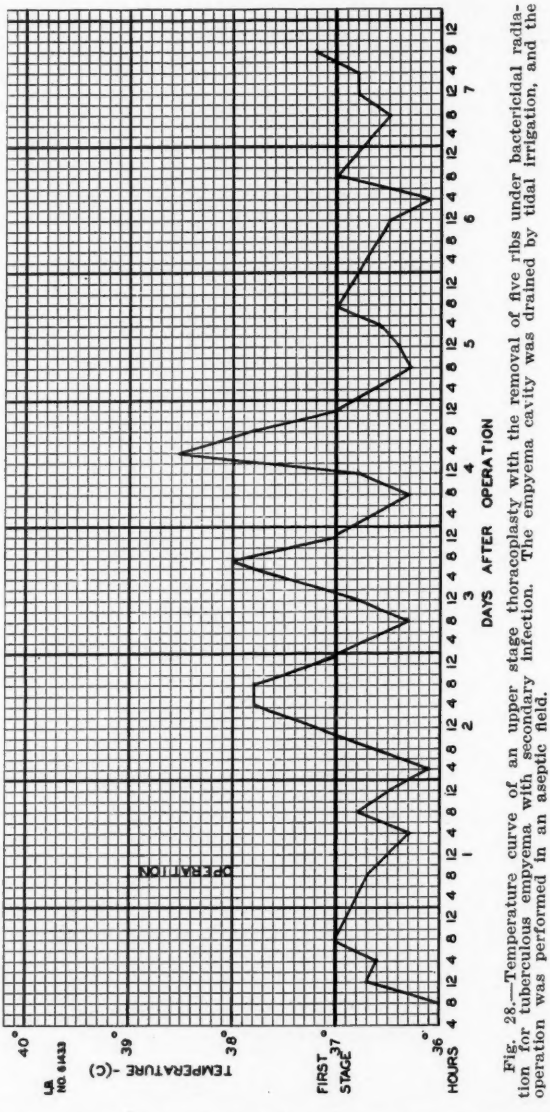


Fig. 28.—Temperature curve of an upper stage thoracoplasty with the removal of five ribs under bactericidal radiation for tuberculous empyema with secondary infection. The empyema cavity was drained by tidal irrigation, and the operation was performed in an aseptic field.

formed from January to April, and that during part of that time there was a severe epidemic of influenza and certain surgeons on the staff discontinued all operating except for emergencies. In the four preced-

ing years, because of the fear of infection, we had performed only ten thoracoplasties during the months of January, February, March, and April.

Since beginning the use of bactericidal radiation we have modified our technic somewhat. No drains are inserted. The second operation is frequently performed through the scar of the first (Fig. 15), since

TABLE IV

TIME REQUIRED FOR HEALING

The figures in this table must be interpreted in view of the fact that most of the operative wounds performed without the radiation were drained, while only two of 18 of those performed with the radiation were drained. These two healed within an average of eleven days, as compared with an average of twenty-one days for the uninfected cases operated on without the radiation. One must also remember that all the cases operated on without the bactericidal radiation and recorded as uninfected had a bacterial contamination and the drains were inserted because of the fear of infection. If there were no question of life and death, or pain and discomfort, or smooth as contrasted to stormy convalescence, the saving to the hospital and the patient in time of hospitalization alone would more than justify the expenditure necessary for installation of these tubes.

	WITHOUT RADIATION DAYS	WITH RADIATION DAYS
Uninfected Cases		
Average duration	21	9
Shortest duration	11	7
Longest duration	44	30*
Infected Cases		
Average duration	34	8†
Shortest duration	16	8†
Longest duration	73	8†

*Uninfected wound separated on the eighth day as a result of undue tension.

†Culture of *Staphylococcus albus* from about a suture in one case. Healing was not delayed.

we feel reasonably certain that there are no latent organisms in the partly healed wound. The operative stages are brought closer together with a saving of time to the hospital and the patient.

SUMMARY

Tubes designed to produce controlled intensities of the most effective bactericidal radiation are introduced to eliminate air-borne contamination of operative wounds. This radiation kills practically all bacteria in the air within a radius of eight feet within ten minutes and kills a sprayed culture of hemolytic *Staphylococcus aureus* at five feet within sixty seconds.

Eight tubes, as illustrated in Fig. 8, do not completely sterilize, within an hour's exposure, the outlying parts of an operating room 20×20 feet in size when it is occupied. We are planning to sterilize the air of the entire room with a wider distribution of radiation tubes. A

barrage of radiation is laid down which kills practically all bacteria in the air before they can reach the wound which is beneath the center of the cluster of tubes.

This radiation from eight tubes at five feet distance produces, on a blond, only an erythema after eighty minutes exposure. Wounds in rats and dogs exposed at this distance for thirty minutes and ninety minutes respectively heal as well as, or better than, similar wounds in the control animals.

The incoming air in our operating rooms is practically sterile. These radiation tubes are made necessary by the following conditions:

A. The nose and throat of a high percentage of the population are at times contaminated if not actually infected with the *Staphylococcus aureus* or other pathogenic bacteria, and masks will not prevent the contamination of the air.

B. Wherever there are human beings, the air becomes contaminated despite all precautions that we use.

C. Wounds are more likely infected from the air where there are large numbers of these pathogenic organisms than from the skin of the patient or hands of the operating team, which, when properly cleaned, are practically sterile.

D. We have been unable to eliminate completely these bacteria from the air of the operating rooms by any other means at our disposal. They have been reduced 60 per cent to 90 per cent by the following measures:

a. Masks worn *at all times.*
b. All known chronic carriers of pathogenic bacteria kept out of the operating rooms at all times.
c. The number of individuals in the operating rooms reduced to the minimum. Visitors and students not allowed.
d. The rooms painted frequently and washed daily.
e. By forced ventilation the contaminated air replaced by clean air taken from above the roof and washed.
f. Major operations performed before the room had been long occupied.

E. This reduction has not been sufficiently great to eliminate all infections. In all infected cases in which cultures of the air had been made during the operation the organism obtained from the wound was the same as the one cultured from the air.

F. Daily cultures of the air showed the danger periods for large operations, but these could not be eliminated.

The efficiency of the radiation from these tubes in sterilizing the air is well demonstrated by incubated culture plates exposed with and without the radiation. Practically no organisms grew on the cultures exposed within eight feet of the tubes with the radiation on, as contrasted to the large number present under other conditions.

Clinically the results in 18 thoracoplasties performed in a field of bactericidal radiation as contrasted to 110 thoracoplasties performed without this radiation is proof of their efficacy.

A. Deaths from infected wounds	with radiation	0
	without radiation	4
B. Infected wounds	with radiation	0%
	without radiation	33%
C. Postoperative temperature elevation,		
Average highest	with radiation	37.5° C.
	without radiation	38.5° C.
38° C. or below	with radiation	72%
	without radiation	32%
Average duration in days	with radiation	2
	without radiation	9
For four days or longer	with radiation	33%
	without radiation	87%

All patients operated on under the radiation from these tubes had an unusually smooth convalescence. In addition to having no infection and a lower and shorter period of temperature elevation, there was less pain, more rapid healing, and quicker return to the preoperative general condition.

The morale of the staff has been greatly improved by restoring their confidence in the sterile surroundings for the operative work.

CONCLUSIONS

In the present stage of surgery we believe that pathogenic bacteria given off by human beings and floating in the air cause the major portion of infected wounds originating in the operating room.

We believe that the special bactericidal radiation presented here will eliminate this hazard of infection and that it is the only practical means available at the present time.

As surgery has embraced operative procedures of greater magnitude and with inevitable trauma and as sterilization of everything but the air has become more satisfactory, this danger of air-borne bacteria, once of little significance, has now assumed a major rôle. The concentration of operative work and more continuous utilization of operating room space has added to this hazard by increasing the pollution of the air. With large numbers of visitors the air contamination must be increased. The

old operators working on the kitchen table may have had poorer sterilizing facilities, but the air of the operative region was probably cleaner than in most of the operating rooms of today.

We are convinced that in our operating rooms the greater number of staphylococci which cause infections reach the wound by way of the air rather than by skin contamination. We do not advocate giving up any steps in skin sterilization technic, but urge each surgeon to consider critically whether or not the infection comes from the air or the skin.

We believe that without sterilization of the air, as by this use of bactericidal radiation, every operative wound is contaminated, which means that infection is present. At times these invaders are quickly killed or walled off by the host, while at other times they may go on to suppuration or even cause the death of the host.

We believe that the postoperative temperature rise of the first few days is in most cases caused more by this bacterial contamination than by the absorption of blood and traumatized tissues. This statement does not take into consideration the reaction from compressing a tuberculous cavity which is infected, but it is my opinion that even in these cases at times the temperature ascribed to this phenomenon may be caused more by bacterial contamination of the operative wound than by absorption of tuberculin products from the lesion in the lung.

Already for any large operation we feel that we should insist on sterile air in the operative field just as we demand sterile supplies and instruments. The development of a relatively atraumatic surgical technic should be the aim of every surgeon or teacher of surgery. We believe, however, that the day is at hand when surgeons are no longer justified in depending on this to keep the bacteria which fall in a wound from gaining a foothold. Instead, the bacteria should be eliminated from the air or killed before they can enter the wound. We are no longer justified in taking any chance that the organisms may have more resistance than the host even in the patient having a smaller incision with the minimal trauma.

The number of infected wounds is still high (10 per cent to 20 per cent) as indicated by reports from certain clinics in which a careful survey has been made and accurate records have been kept. The majority of these are so mild that the profession and public have not become aroused. I believe that these can be greatly reduced by bactericidal radiation and that soon the public will no longer consider the infection beyond the control of the surgeon. Already we operate on clean cases without great fear of infection, whereas formerly the dread of bacterial contamination from an uncontrollable source was ever present.

REFERENCES

Blatt, Maurice L., and Dale: A Bacteriological Study of the Efficiency of Face Masks, Surg. Gynec. Obst. 57: 363, 1933.

Brewer, G. E.: Studies in Aseptic Technique, J. A. M. A. 64: 1369, 1915.

Capps, J. A.: Measures for the Prevention and Control of Respiratory Infections in Military Camps, J. A. M. A. **71**: 448, 1918.

Dannenberg, A. M.: A Simple Face Mask for Use by Contagious Disease Attendants, J. A. M. A. **70**: 990, 1918.

Dandy, W. E.: The Importance of More Adequate Sterilization Processes in Hospitals, Bull. Am. Coll. Surg. **16**: 11, 1932.

Davis, John Staige: The Importance of Adequate Masking During Operations, Ann. Surg. **100**: 1008, 1934.

Doust, B. C., and Lyon, A. B.: Face Masks Infections of the Respiratory Tract, J. A. M. A. **71**: 1216, 1918.

Goff, B. H.: Analysis of Wound Union in 3,000 Abdominal Incisions Based on Woman's Hospital Classification of Wounds and Wound Union, Surg. Gynec. Obst. **41**: 728, 1925.

Halsted, W. S.: Ligature and Suture Material. The Employment of Fine Silk in Preference to Catgut, J. A. M. A. **60**: 1119, 1913.

Hunt, Ernest L.: Some Further Observations Upon Contamination of Operative Wounds (Air Borne Bacteria), New England J. Med. **209**: 931, 1933.

Kellogg, W. H., and McMillen, G.: An Experimental Study of the Efficiency of Gauze Face Masks, Am. J. Pub. Health **10**: 34, 1920.

Leete, H. M.: Some Experiments on Masks, The Lancet, p. 392, March 8, 1919.

Meleney, F. L., Humphreys, F. B., and Carp, L.: Unusual Fatal Operative Wound Infection Yielding a Pathogenic Anaerobe of Gas Gangrene Group Not Hitherto Described With Direct Reference to Catgut as the Source, Surg. Gynec. Obst. **45**: 775, 1927.

Meleney, F. L.: Infection in Clean Wounds, Surg. Gynec. Obst. **60**: 264, 1935.

Meleney, F. L.: Seasonal Incidence of Hemolytic Streptococcus in Nose and Throat, J. A. M. A. **88**: 1392, 1927.

Meleney, F. L., and Stevens, F. A.: Postoperative Hemolytic Streptococcus Wound Infections and Their Relation to Hemolytic Streptococcus Carriers Among the Operating Personnel, Surg. Gynec. Obst. **43**: 338, 1926.

Mellinger, H. V.: A New Mask That Protects Both Physician and Patient, J. A. M. A. **95**: 662, 1930.

Vivier, P.: The Bacteriology of Celiotomy Wounds Sutured With Catgut and Silk. Part of Thesis Presented to Columbia University for Degree of Master of Medical Science, 1934.

Walker, I. J.: How Can We Determine the Efficiency of the Surgical Mask? Surg. Gynec. Obst. **50**: 266, 1930.

Weaver, G. H.: The Value of the Face Mask and Other Measures, J. A. M. A. **70**: 76, 1918.

Weaver, G. H.: Droplet Infection and Its Prevention by the Face Mask, J. Infect. Dis. **24**: 218, 1919.

Whipple, A. O.: The Use of Silk in the Repair of Clean Wounds, Ann. Surg. **98**: 662, 1933.

Zienkiewicz, Jan: Report of a Case of Meningitis Due to Prolonged Exposure to Direct Rays of the Sun, Pedjatrja polska **14**: 82, 1934. J. Dis. Child. March, 1936, p. 735.

PULMONIC ALVEOLAR EPITHELIUM

A Round Table Conference*

Report of Charles C. Macklin, M.D., Chairman†
London, Canada

THE PROBLEM of the presence or absence of a continuous epithelial lining in the mammalian adult pulmonic alveolar wall and some of its correlations were discussed by five participants.

Dr. J. L. Bremer, of Harvard University, dealt particularly with the growth and regeneration of lung alveoli and their bearing on the problem of the alveolar lining. He conceived of the alveoli as being lined by a continuous epithelial layer of entodermal origin whose unit is a cell with a relatively thick body from which a thin flange or film extends over the pulmonary capillary. This cell body and its process rest upon a thin layer of connective tissue. The epithelial layer is thus a persistance (with differentiation) of the embryonic condition. It is admitted that the flange is invisible by ordinary histologic methods; but so, too, for instance, are some of the living protoplasmic films of tissue cultures, and the epithelium of the visceral layer of Bowman's capsule in the kidney. The flange is contractile and adapts itself to the area variation of the alveolar wall which it covers. It may be retracted after death, or in fixation, whereupon the capillary appears to be nude. In the budding of new alveoli from old in later fetal and early postnatal life a lining epithelium is necessary; indeed the continuous formation of alveolar buds in the postnatal lung argues strongly for the continuity of the alveolar epithelium. In the conversion of air sacs to bronchioles, in development of the lung, the epithelium changes from "respiratory" to cuboidal. The epithelium may be well studied in developing opossum lungs, which provide favorable material for viewing epithelial flanges over capillaries. Epithelium is essential to lung repair. If no epithelium is present, what is there to confine the tissue fluid? Terry has described a fluid film on the alveolar wall of the frog (Dem. No. D57 of the present congress), but this is distinguished from ordinary tissue fluid by being uncoagulable. The alveolar phagocytes may be of entodermal origin, though the origin from mesoderm of some of them is not ruled out.

Microscopic preparations were demonstrated by Dr. Bremer.

*This Conference was held at Duke University, Durham, N. C., on April 11, 1936, from 9 A.M. to 12 noon, under the auspices of the American Association of Anatomists, Fifty-Second Session, Warren H. Lewis, President.

†From the University of Western Ontario.

Received for publication, July 13, 1936.

Dr. Dwight M. Palmer, of Ohio State University, based his conclusions on sections, often thick by preference, from good human fetal material fixed while still warm. Although his observations confirmed the view that in the earlier stages the future alveoli have a continuous epithelial lining, he found that during the twenty-first week of fetal life the epithelium of the future alveolus underwent a profound change, the weakly staining cytoplasm becoming loaded with birefringent lipoid material, while the nuclei appeared vesicular; and at the same time young capillary loops pushed in between the changed epithelial cells and displaced them. These capillary loops now appeared to be devoid of epithelium and formed part of the lining of the alveolus. He further pointed out that the embryonic lung was composed predominantly of mesenchyme. This element made up two-thirds of the entire mass in the twenty-first week of development, while the entodermal epithelium contributed only one-third. His testimony thus went to support the view that there is not a continuous epithelium on the alveolar wall. Non-nucleated plates were not found.

Dr. William Bloom, of the University of Chicago, reported that he had been unable to find irrefutable evidence of an epithelial lining in the adult alveolus and regarded the preparations of others, adduced in support of epithelium, and particularly those involving the silver nitrate technics, as unreliable or capable of other interpretation. It is admitted that there is a continuous epithelium in the alveoli of cold blooded animals and that there is no epithelium in birds. In his view, the epithelium of the future alveolus becomes discontinuous in fetal life, and the blood vessels push out and line the walls of the future air spaces. Most of the remaining epithelial cells disappear before or at birth, but there is no evidence that all of the epithelium disappears. The alveolar phagocytes may be of dual origin—entodermal as well as mesodermal. Some might be of hematogenous origin. Dr. Bloom feels that no one has unequivocally demonstrated epithelium in the alveolar wall by silver nitrate in warm blooded animals, though it may be done in the frog and turtle. If there is really an epithelial layer or flange present in the mammalian alveolus it could be demonstrated histologically by modern methods, for this layer is admittedly very thin in the frog and turtle, for instance, and yet can be made clear to the eye. Mesenchymal derivatives may assume epithelial arrangements—as in tissue cultures—and what has been interpreted as an epithelial layer separated from the underlying alveolar wall, in pathologic conditions, may be composed of mesodermal cells and exudate. There is no cuboidal or flattened epithelium visible in the alveoli of the collapsed lungs of experimental pneumothorax, and it should be visible if present.

Dr. Bloom demonstrated microscopic slides prepared by Dr. Loosli showing a mononuclear phagocyte apparently in the act of passing through an alveolar pore, and strands of fibrin linking neighboring

alveoli through an alveolar pore. Pores are normally present and easily demonstrable, and their existence is in favor of the absence of a continuous epithelial lining.

At this point there was a natural break in the program between the consideration of the epithelial problem in the normal lung, on the one hand, and in the pathologic lung, on the other. Because of this, opportunity was given for a free exchange of views among the various participants, who had already spoken, regarding evidence for and against the presence of epithelium in the normal mammalian lung. Dr. Bremer could not visualize an alveolus with unprotected, raw surfaces, which is yet capable of postnatal regulated growth by budding. Dr. Palmer made it clear that he was arguing against the view of a *continuous* epithelium, rather than that of a total absence of epithelium, and stated that he had been able to demonstrate to his satisfaction that the previously intact epithelial lining becomes discontinuous at the twenty-first week of fetal development; but he was not prepared to say what happens to all of the old epithelial cells. Dr. Bloom reaffirmed his previously stated views.

The conference then proceeded to take up the findings bearing on the problem in the pathologic lung.

Dr. Douglas H. Sprunt, of the Department of Pathology of Duke University, discussed the response of the alveolar epithelium to infection. He pointed out that the lining cells of the alveoli, while difficult to see in normal lungs, became easily apparent in certain morbid conditions, and that they then presented the morphology and staining characteristics of epithelium. He averred that in this condition they had no phagocytic power. He showed lantern slides from photomicrographs taken from experimentally altered animals as well as from human necropsy material, giving examples of the alveoli lined by a layer of epithelioid cells. An epithelioid proliferation was produced by the viruses; namely, psittacosis, epidemic influenza, and vaccinia; by toxins; by the Bordet-Gengou bacillus (pertussis); during the healing stage of pneumococcus pneumonia; by congenital syphilis; by chronic passive congestion; and as a result of chemical irritation. It is important to choose only those animals in which the dose of the incitant has been sufficient only to stimulate the epithelioid cells to proliferation, for if it has gone beyond this stage and has engendered necrosis, then fibrosis ensues, and in this case the changes are very difficult to interpret.

Dr. Sprunt explains that with the common bacterial pneumonias there is a predominance of the polymorphonuclear cells in the inflammatory reaction, and, since the patient or animal usually dies at the height of this reaction, little if any attention has been paid to the changes in the epithelial tissues. In the virus infections, however, the polymorphonuclear reaction is only transient, lasting at best not over one or

two days, and the animals or patients die at the height of the epithelial reaction, and thus this type of reaction is best for studying the epithelium.

Dr. Sprunt finds frequently in these pathological cases, both in the pulmonic interstitial tissue and in the alveoli, mononuclear cells of two types; namely, phagocytic and nonphagocytic; and he feels that the nonphagocytic cells are in many instances epithelial.

He thus feels that additional proof of the epithelial nature of the alveolar lining cells is given by the reaction of the lining cells to stimulation by viruses, toxins and chemicals.

Dr. B. M. Fried, of New York, interpreted his material, of wide variety, from experimental animals and human subjects, as failing to demonstrate an epithelial lining. He regarded the large mononuclear phagocytes of the alveoli, and the exudate cells of inflammation and congestion ("dust" or "heart disease" cells) as of mesodermal origin exclusively and as developing only from the alveolar septums. In the early stages of the disease they are produced locally and not imported from outside sources. He thus rules out origination of these cells from the circulating blood, the endothelial cells lining the pulmonary capillaries, or from the local lymphoid tissue. In normal conditions the wall of the air sacs contains a scant number of undifferentiated mesodermal cells embedded in the septal reticulum. The septums are lined not by a syncytial row of cells but by scattered cells which are particularly noticeable at the bifurcation of the capillaries. In inflammatory and congestive conditions the cells present in and on the septums behave like mesenchymal cells found elsewhere in the body, rapidly increasing in size, proliferating with an astonishing promptness, wandering out into the alveolar lumen, and exhibiting strong ameboid phagocytic activities.

While these traits are noticeable in any inflammatory or congestive disease of the lung, they are particularly conspicuous when a proper antigen is introduced into the lungs via the trachea. The tubercle bacillus and lipoid substances, respectively, which Dr. Fried has used, among other agents, are probably the most suitable materials. The cells lying in and on the septums are those that become activated by Koch's bacillus and by the fatty material, respectively. They promptly phagocytose the acid-fast bacilli; they participate in the formation of the tubercle; they form giant cells of the Langhans type; and they form the elements of the tuberculous granulation tissue. The fat is taken up as fine and coarse granules and is deposited in their cytoplasm. The cells engorged with fat form typical lipoid granulomas resembling xanthomatous nodules.

Thus the cells of the pulmonary septums, i.e., the so-called "alveolar epithelial cells," are concerned in the defensive and metabolic processes in which the lungs are involved; they act in ways similar to the Kupffer

cells, the splenocytes, and the reticular cells of the bone marrow and the lymph nodes. In brief, the physiologic and morphologic characteristics indicate that they are in all likelihood of mesodermal origin.

It is most interesting that the so-called alveolar epithelium, which, as all agree, is so sensitive to stimuli, has never been observed to give rise to an epithelial malignant disease. The opinion is prevalent (or quasi-unanimous) that cancer originating primarily in the lungs is of bronchial and not of alveolar origin.

Dr. Fried showed numerous lantern slides in support of his views.

A free discussion among the participants then took place, in which specific questions were asked and answered and the views of each made clear to the others. Three main possibilities were discussed (1) a complete epithelial lining, invisible though it may be with our present methods; (2) an incomplete covering of the wall by surviving entodermal cells; and (3) a wall completely divested of the original epithelium, and composed only of cells of mesodermal origin. It seemed difficult to rule out the survival of at least some of the original epithelium, albeit in modified form. Some of the macrophages might owe their existence to that source.

The discussion was then opened to the audience, and a number took advantage of the opportunity to express their views. Miss Christianna Smith emphasized the glandular pattern of the early lung and visualized the alveoli as an end-result of a continuous developmental process in which an original intact lining epithelium was conspicuous. Dr. Edward McCrady, Jr., pointed out that, although in most mammals there was a change in the developing alveolar wall at or before birth which results in either the elimination or extreme attenuation of the original lining so that it becomes undemonstrable, there was an exception in the case of at least one mammal, for in the opossum a functional respiratory epithelium is clearly demonstrable for some time after birth, although the alveolar lining in the adult opossum is no more demonstrable than it is in any other adult mammal. He has found that the opossum, during most of the period of occupation of the pouch, is poikilothermal. Dr. McCrady does not feel that the distinction proposed for entodermal as against mesodermal cells, namely, that entodermal cells do not ordinarily phagocytose, whereas mesodermal cells do, helps us a great deal; for in certain animals, for instance the coelenterates, it is clear that entodermal cells feed in a typically ameboid fashion by the extension of pseudopodia around particles in the enteric cavity. He remarked that when the adult mammalian alveolar wall was lined with easily visible apparently phagocytic cells the condition was admittedly abnormal, occurring only in diseased lungs; and suggested that the viewing of these as modified entodermal cells might have some encouragement from the observation that in tissue cultures cells very often

de-differentiate and revert to primitive behavior of which they are incapable in their normal environment in the body.

Regarding the observation of the Chairman that, if there is a complete entodermal lining in the alveolus, then the macrophages which are seen in the lumen are really outside the body, whereas, if there is no lining, the alveoli are only tissue spaces and the macrophages within them only normal residents, Dr. McCrady wondered if it is any less difficult to imagine tissue spaces in permanent normal communication with the outside world than it is to imagine certain ameboid cells crawling out on the surface of the body; for in the pelecypod molluscs, ameboid cells from the blood crawl completely outside the body into the mantle cavity where for some time they move about not even in contact with the body, and later they reenter the blood stream through the gills. Also, in human beings, leucocytes normally crawl out into open wounds, and even in the absence of wounds they crawl about the nasopharynx and mouth. Dr. McCrady was careful to add that in the present state of our knowledge it is necessary to withhold judgment on the question as to whether there is or is not an entodermal epithelium in the alveoli of normal adult mammalian lungs. Opportunity was then given for viewing the demonstrations and conferring privately with the participants.

The unavoidable absence of Dr. William Snow Miller, the Dean of Pneumonologists, was regretted by all. He was to have spoken on "The Demonstration of Alveolar Epithelium in the A c u t e l y Pneumonic Lung." His keen interest in the conference, however, was shown by a letter in which he enclosed two excellent photomicrographs. One of these demonstrated, in his view, the stripping of a layer of epithelium from the mesenchymal alveolar wall, including the capillary surface, by an accumulation of fluid exudate, in a case of pulmonary edema. In the other case, one of lobar pneumonia, a section from just beneath the thickened pleura shows, in the opinion of Professor Miller, a layer of epithelial cells covering the walls of the alveoli. He says: "No pathological process will produce the epithelium lining the alveoli." Although his letter arrived too late to be read at the conference, it was read by the participants afterward.

Dr. L. E. Josselyn, of Highwood, Ill., was also regrettably unable to be present, but his views, derived from work done by him at the University of Chicago, and expressed in a letter to the Chairman, may be recorded here. He has been unable to demonstrate in experimental animals the presence of respiratory alveolar epithelium and cannot convince himself of the reliability of the microscopic evidence held by others to support the epithelial hypothesis. He is open to conviction if satisfactory evidence of the presence of epithelium is advanced.

Everyone was sorry, too, that the others on the original tentative program, Dr. Sylvia H. Bensley and Dr. Robert D. Bensley, of the Uni-

versity of Chicago, and Dr. E. V. Cowdry, of Washington University, could not be present.

Though no conclusions were drawn by the Conference, it is hoped that the focusing of attention upon the points at issue will have the effect of stimulating effort which will result in the bringing out of new evidence. For instance, if there is no alveolar epithelium there must be a region, on the airway, where the epithelium of the bronchial tree comes to an end. There must be a "free edge" somewhere. Future research may disclose the position and nature of this border, if it exists. The old idea of a nonnucleated "plate" as a component of the alveolar lining is disappearing. It seems obvious that, before the conception of a continuous epithelial layer for the adult mammalian pulmonary chambers, reasonable though this may seem from the ontogenetic and phylogenetic viewpoints, can have universal acceptance, there must be brought forward evidence of a more convincing and unequivocal character. Apparently the number of those who deny the presence of this epithelium on the ground of their inability to demonstrate it is increasing. The rise of this view to predominance would mean the recasting of our physiologic and pathologic thinking on the subject of the alveolus, which would become a sort of physiologic tissue air space—a "functional interstitial emphysema" in essence. Exudates in the alveoli would be technically inside of the body; reparative processes in the alveolar wall would be simplified by the implication of only one germ layer, the mesoderm; and the problem of the relining of the wall would be eliminated, etc.

MEDIASTINAL LIPOMAS

R. Milnes Walker, M.S., F.R.C.S.
Wolverhampton, England

THE literature concerning fatty tumors in the mediastinum has been reviewed as recently as 1930,[1] and a survey of the pathology of the subject was made by Graham and Wiese,[2] when they recorded a case in 1928; since these papers were written, reports of three further examples of this type of tumor in this unusual situation have been found in the medical literature,[3, 4] which, with the case here recorded, brings the total number of cases to sixteen. In addition to these there are accounts of a few examples of subpleural lipomas arising under the parietal pleura of the chest wall, but it appears that the majority of intrathoracic lipomas commence their growth in the fatty tissues of the mediastinum, and it is only with these that this article deals. No useful purpose would be served in again summarizing the literature of the subject, but an account of the present case is given because it is the first example recorded in detail of a surgical attempt to remove one of those massive mediastinal lipomas which fill a large proportion of the chest cavity, and it is felt that the lessons learned from this case may be of use to any surgeon who is called upon to deal with another similar tumor.

These mediastinal tumors may be divided into two groups from the clinical standpoint, those which have grown out of the chest so that they are partially extrathoracic, and those which are still entirely enclosed inside the chest cavity. It appears that those which arise in the superior mediastinum have a tendency to enlarge upward into the neck, while those which take their origin in the anterior mediastinum may penetrate through an intercostal space, or, as in one example, through a defect in the sternum, and so come to lie partly under the subcutaneous tissues and muscles which cover the chest wall. Those which do not follow one of these courses, but continue to grow inside the chest, gradually encroach on one or both pleural cavities, and may attain a very large size before they cause any symptoms; the largest of these weighed 17 lb. 6 oz. when removed postmortem, and others are described as being the size of a fetal head, or filling a quart cup. The tumor in the case described here must have weighed in the neighborhood of 5 lb. before the first portion was removed.

In the first group of cases, those in which the tumor was extending into the base of the neck, there are two examples, those of Beatson,[5] and of Graham and Wiese[2]; both of these were successfully operated upon, the

Received for publication, June 29, 1936.

size of the tumors being "an orange," and "twice a hen's egg" respectively. In the group which were partly extrathoracic because they penetrated the chest wall, there are four cases; in two, those of Cruveilhier[6] and Krönlein,[7] the external portion alone was removed, but the patient succumbed; the third, that of Garnier and Grosjean,[8] survived a similar operation; while the fourth, the case of Beyers,[9] in which the intra- and extrathoracic portions were each two inches in diameter, recovered after successful removal of the whole tumor.

It is the totally intrathoracic group to which the present case belongs; there are nine previous records of such tumors; in six, those of Fitz,[10] Ewing,[11] Bertoli,[12] Leopold,[13] Lemon[14] and Yater and Lyddane,[1] the nature of the tumor was definitely established only on postmortem examination, the patient having died of pulmonary complications; in Lemon's case, however, the true nature was strongly suspected during life, the patient having other lipomas, one in each supraclavicular fossa of the neck, unconnected with the mediastinal tumor; in the seventh, the case of Schinz and Gasser,[3] the tumor was thought to be a lymphosarcoma, occupying the upper part of the right pleural cavity, and was explored by operation, but the patient succumbed, apparently before the tumor had been reached, because the report states that "at the postmortem examination the tumor was found to be a lipoma." The eighth and ninth cases are described briefly in the Report of the Chest Tumor Registry.[4] In both these cases surgical removal was attempted; the first patient died during novocaine infiltration before the operation was commenced, the tumor being found at autopsy to measure $25 \times 25 \times 13$ cm.; in the other, the tumor was partly removed successfully, but as in the case here described, it rapidly recurred and the patient succumbed, no further operative treatment being attempted. Up to now therefore, there is no record of a completely intrathoracic lipoma having been successfully removed.

CASE REPORT

The patient was a boy aged thirteen years, whom I was asked to see on September 27, 1935, by Doctors H. A. Johnson and S. C. Dyke. Up to the present illness he had always been a healthy child. There was a history of his losing weight for the last three months, during which time a whistling noise had been noted as he breathed during sleep, but this did not occur when he was awake. During the last fortnight it had been noticed that he had been getting short of breath when he took exercise. There was no cough and his appetite was good. He was a thin boy but with well-developed bones. While at rest his respirations were 24; there was active movement of the alae nasi and slight cyanosis. On examination of the chest there was some bulging observed over the second, third, and fourth right intercostal spaces in front; the heart apex beat was in the seventh space, 11.5 cm. to the left of the midline. There was very poor expansion of the right side of the chest; the whole of this side was dull to percussion except a small area in the axilla. Cardiac dullness was continuous with this dull area as far as the apex beat. Breath sounds over the whole of the right side of the chest were bronchial in type except for the resonant area

in the axilla where they were vesicular. There were normal breath sounds over the left lung, and no distended veins were observed over the chest or neck. Abdominal examination was normal.

A puncture had been performed the previous day by Dr. Dyke in the eighth space posteriorly, but no fluid obtained, the needle sinking into a soft mass without response. A radiogram of the chest showed a large, rounded shadow occupying almost the whole right chest, except for a triangular area above the diaphragm in the axilla; this swelling extended across the midline and had a smooth rounded border 5 cm. to the left of the midline overlapping the aortic shadow. The heart was displaced to the left. (Fig. 1.)

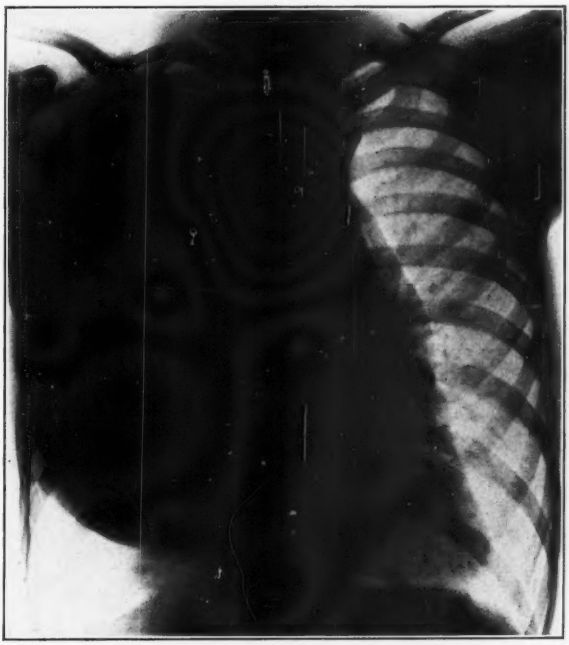


Fig. 1.—Before first operation. Note the lobulated margin of the tumor.

A puncture was performed in the third intercostal space in the midclavicular line. After piercing what felt like a firm layer of tissue the needle entered a soft mass without resistance, "which felt like butter." No blood or other fluid was obtained on aspiration. A diagnosis of mediastinal dermoid was made, and it was proposed to explore this with a view to marsupializing it as in the case of teratoma reported by Morriston Davies.[15]

Operation was performed on October 4, 1935, gas and oxygen being administered by Dr. Winifred Dean, through a face piece under pressure, without intratracheal intubation. An incision was made in the fourth intercostal space from the axillary line anteriorly, turning up over the third costal cartilage. The pleura which was now exposed, was opened and a large yellowish tumor was seen projecting from the mediastinum, being covered with pleura; there was no fluid in the pleural cavity, and the lung was compressed to the size of a fist and lying posteriorly, extending as

far as the axilla. The tumor was definitely lobulated and quite soft. The mediastinal pleura over the tumor was now sutured to the parietal pleura, so closing the right pleural cavity. The pleura which formed a capsule to the tumor was then incised, and a large lobule of the tumor, measuring 18.5 × 8 × 7 cm., and weighing 515 gm., and which was passing obliquely upward and to the left behind the sternum, was enucleated without much difficulty. A small drainage tube was now inserted and the wound closed round it. Intravenous saline was given during the operation.

Three hours later a considerable oozing began to occur from the tube, and his pulse rate was rising, so a 500 c.c. blood transfusion was administered. During the subsequent twelve hours much anxiety was caused owing to the continued oozing from the tube and to a recurrence of the severe stridor. The question of reopening the wound and packing it was carefully considered, but it was determined that his con-

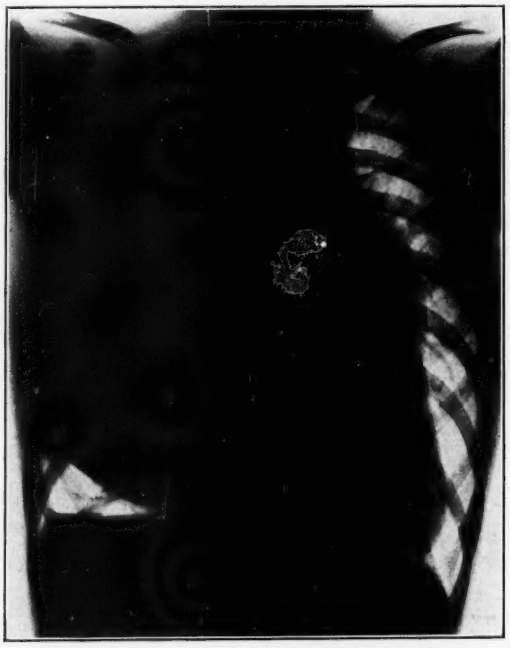


Fig. 2.—Before second operation. Note the increase in size of the tumor in six months in spite of the removal of a lobe weighing 515 gm.

dition was unfit to stand this, and it was thought wisest not to touch the wound. His pulse had considerably improved after the blood transfusion and remained fairly good though rapid. Following injection of atropine 1/100 grain the stridor became less, this evidently being due partly to edema of the trachea and accumulation of secretion.

Twelve hours after the operation his condition quite definitely began to improve, and a few hours later his breathing was easy, his color good, and his pulse improving. There was no further oozing from the wound. The tube was removed on the fourth day, and the stitches were removed four days later. A fortnight after the operation he began to get up, and went home on the twenty-second day.

During the convalescence the whole of the right side of the chest remained dull to percussion, and the breath sounds were poor, suggesting the presence of fluid in

the right pleural cavity, but as he was showing steady improvement the chest was not aspirated. It was proposed to watch any progress of the tumor by regular radiograph examinations and wait until the following summer, with a view to re-opening the chest then and removing part or all of the remainder of the tumor. Three months after the operation his condition was remaining good. He could run about in moderation without getting breathless, and there was no stridor. There was still dullness to percussion over the front of the right chest and axilla, but a less impaired note all down the back of the chest. The position of the heart apex beat had, however, remained unchanged. Radiographic examination at this time showed a very similar appearance to the previous examination; the outline of the tumor was no smaller, but in view of the fact that the lobule that had been removed was lying anteriorly, this did not arouse suspicion that it was growing again rapidly.

He was not seen again until April 4, 1936, when he had been getting considerably more breathless for the last week. This was much worse during the last two days,

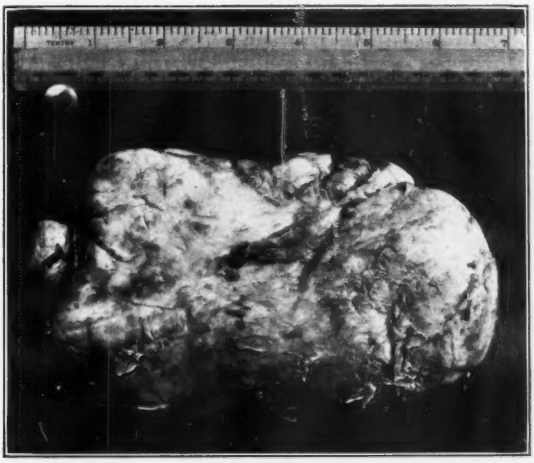


Fig. 3.—Portion of tumor removed at the first operation.

and the whistling at night had recurred. He was then short of breath even while sitting in bed. There was a dull percussion note over the whole of the right of the chest, and the position of the left border of the heart was still the same. A radiogram showed that the whole of the right chest was occupied by the tumor, except a small area in the costophrenic angle, and the tumor had spread farther across the midline above the heart. The trachea was displaced to the left, so that its intrathoracic portion was running very obliquely. (Fig. 2.)

In view of his distress it was decided that operation should not be delayed. A half dose of avertin was given by rectum; following this he showed signs of respiratory distress and became very cyanosed, but this was relieved to some extent by the passage of an intratracheal catheter. Gas and oxygen was then administered under pressure through this by Dr. Winifred Dean. The old wound was reopened and the capsule of the lipoma incised. It was then found that the tumor was breaking down and was partly liquefied. Fat could be easily removed from inside the tumor by the handful in some places; some fat was adherent to the capsule, but most of it was easily detached and removed. Altogether in this way a quantity of

fat weighing 1.47 kg. was removed, this forming practically all the tumor, though some fat was left adherent to the capsule. The residual cavity occupied most of the right chest and spread across the midline behind the sternum in front of the trachea. In view of the trouble with oozing which had followed the previous operation, a large gauze roll was inserted, and this was brought out through a tube which was tightly plugged, and the wound closed to make it airtight. Blood transfusion had been given during the operation.

His general condition had started to improve as soon as the volume of fat in the chest was reduced, and at the end of the operation his condition was fairly good. Continuous intravenous saline was administered, and his condition remained satisfactory for three hours, after which time his color rapidly became more cyanosed. During the following twelve hours his color remained poor in spite of administration of oxygen, but the pulse volume was fair. He was able to talk and take liquids by mouth, though there was some stridor present. Twenty-one hours after the operation he suddenly collapsed, and though respirations continued for a few minutes the heart had apparently stopped. Attempts at reinflating his lungs with oxygen failed, and he showed no sign of recovery. The plug was removed from the wound and the cavity was quite dry, there being no effusion into it. No postmortem examination was made.

It seems probable that death was finally due to cardiac embarrassment from the severely displaced mediastinum, though this was undoubtedly aggravated by the anoxemia, which was probably due to failure of expansion of the right lung, and edema of or pressure on the left bronchus.

Andrus[16] remarks in the Report of the Chest Tumor Registry that "A striking and encouraging feature of certain of these tumors reported in the literature is just this fact, i.e., that even when large, their enucleation, considering their size, would appear to be relatively much more easy of accomplishment than is the case with other and smaller tumors."

It may be easy for the pathologist to remove one of these massive tumors in the postmortem room, but the problem in the operating theater is quite different; the tumor may shell out without any difficulty, but it is a very much more serious problem to deal with the changes in the mechanical conditions inside the chest cavity which result from such an operation; during the time which the tumor has been slowly increasing in size the heart and lungs have become gradually accommodated to the strains and pressures which the large tumor puts upon them, and the removal of the tumor results in equally great stresses being suddenly put upon these organs. The difficult questions to be settled are:

1. The large size of the tumor, and consequent small amount of available expansion of the lungs leads to difficulty in connection with the anesthetic; the anoxemia following a very small dose of avertin may result in very serious collapse.

2. It may be difficult to control the oozing which is likely to occur from the flimsy capsule when the tumor has been enucleated; the negative pressure inside the chest and the movements of the chest wall and mediastinum encourage the persistence of this oozing.

3. The displacement of the mediastinum following the removal of the tumor is likely to cause considerable embarrassment of the heart's action.

4. The question of obtaining and maintaining the expansion of the lungs is a difficult one.

It is felt in reviewing the progress of the present case after the second operation that the fatal result was brought about by failure to deal satisfactorily with the last two problems. The first of the difficulties enumerated above was encountered before the second operation, and the second problem was the most serious complication after the earlier operation.

In the light of very meager experience of the present case I venture to put forward a few suggestions as to the means of dealing with these difficulties. As regards the first there is no doubt that the operation must be carried out under positive pressure anesthesia, as one and possibly both pleural cavities may have to be opened; I have used nitrous oxide and oxygen for most major chest operations, often after a preliminary dose of avertin, but there is no doubt that the latter is dangerous when there is marked anoxemia, and no preliminary sedative should be given under such circumstances. I have as yet had no personal experience of cyclopropane, which may prove to have definite advantages over nitrous oxide; in the present case the patient was very anxious about the question of a second anesthetic, and it was in order to overcome this as far as possible that the small dose of avertin was given, but there is no doubt that he would have been better without it.

The second problem is that of oozing from the lining of the cavity; it was in order to prevent this that a gauze roll was inserted at the second operation, and this served its purpose well; what had been the most serious postoperative complication after the first operation did not arise after the second, and when the roll was removed at the time of the patient's death, the cavity was found to be practically dry; it is felt that it would be a wise precaution to insert such a gauze pack, even if it meant a second anesthetic and opening up the wound in order to remove it after some days. In the present case the end of the gauze roll was brought out through a rubber drainage tube, and the tube closed so as not to admit air.

It is felt that it was the dangers brought about by the third problem, the mediastinal displacement, which were responsible for the fatal result which marked the termination of the present case; the heart was displaced far out to the left by the tumor, and the rapid movement of the mediastinum back toward its normal position undoubtedly gave rise to considerable embarrassment of its action; it is considered that this difficulty can only be overcome by not removing too much of the tumor at one time, where the tumor is very large and causing mediastinal displacement as it was in this case. Of course, were the tumor diagnosed earlier before it had reached these dimensions, its complete removal at one stage should not give rise to danger from this source.

In the present case it was hoped that some expansion of the right lung, which had been compressed to a thin layer of tissue occupying the posterior and lower part of the pleural cavity, would be obtained at the time of the operation, using considerable positive pressure with the gas and oxygen apparatus; but though the pressure was raised to 20 mm. Hg before the chest was closed, no expansion of this lung occurred. It appeared that there had been some pleural thickening as a result of the effusion which followed the first operation, and this was the reason the lung remained collapsed; its gradual expansion might have been expected to occur provided a negative pressure had been maintained inside the chest; it is therefore felt that it would be better to close the wound in the chest wall completely, and aspirate later any effusion which forms, rather than leave even a closed drainage with a seal. This would prevent sudden movements of the mediastinum while at the same time maintaining a slight negative pressure in the pleural cavity. If there was any oozing of blood from the lining of the capsule of the lipoma which could not be adequately arrested before the wound was closed, it is suggested that a gauze pack should be lightly inserted, and the wound closed tightly; about the third day, under gas and oxygen anesthesia, part of the wound would have to be reopened for the removal of this pack.

On the clinical side of the question I have only a few comments to make. These tumors are so rare that their occurrence is not likely to be diagnosed definitely except by exploratory thoracotomy, but where the tumor attains a relatively large size it closely resembles a mediastinal dermoid cyst. Both give rise to no symptoms until they have attained a considerable size; there are two points, however, which might lead one to suspect that it was not the more common dermoid cyst with which one was dealing; the radiogram might show that the outline of the tumor was somewhat lobulated, instead of being a regular sphere as is the case with dermoids; and on aspiration of the tumor there is no thick wall as is usual in dermoids, and when the point of the needle is inside the tumor it slips forward without any resistance, yet no fluid, and not even blood escapes from it; in the case of a dermoid the contents are often too thick to be drawn along a needle, but they are likely to offer a little more resistance to the passage of the needle than in a pure fatty tumor. There is one further point of clinical interest; it is the rate at which the tumor increased in size during the six months between the two operations; during this time it had more than made up the 515 gm. which had been removed on the first occasion. The accounts of some of the recorded cases show that in them the tumors took many years to grow, yet it is evident that in other cases the growth may be very rapid.

The question of the treatment of these tumors by other methods apart from their surgical removal has been under consideration. We have

records of three cases in which radiotherapy has been employed, but there is no evidence that this had any beneficial influence in preventing the growth of the tumor. If there were a safe means by which the fat in the center of the tumor could be liquefied sufficiently to be aspirated through a needle or cannula, the tumor might be in this way considerably reduced in size, thus making the subsequent operation for its removal less hazardous; but I am not at present aware of any such means of bringing about the liquefaction of fat in vivo. This suggestion, however, is put forward as a possible means in the future of dealing with large tumors of this nature.

SUMMARY

A case of a large lipoma of the mediastinum is recorded, together with an account of its attempted removal in two stages; the difficulties in dealing with such cases are enumerated and suggestions put forward as to how these can be overcome.

REFERENCES

1. Yater, W. M., and Lyddane, E. S.: Lipoma of Mediastinum, Am. J. M. Sc. **180**: 79, 1930.
2. Graham, E. A., and Wiese, E. R.: Lipomas of the Mediastinum, Arch. Surg. **16**: 380, 1928.
3. Schinz, H. R., and Gasser, E.: Mediastinallipom, Röntgenpraxis **5**: 821, 1933.
4. Andrus, W. DeW.: Lipoma of Mediastinum, Report of Chest Tumor Registry, J. THORACIC SURG. **4**: 241, 1934.
5. Beatson, Dr.: A Fatty Tumour Removed From the Superior Mediastinum, Glasgow M. J. **51**: 57, 1899.
6. Cruveilhier, J.: Traité d'Anatomie Pathologique Générale, **3**: 315, 1856.
7. Krönlein, R. U.: Geschwulst des Mediastinum anticum, Arch. f. klin. Chir. **21**: Suppl., 157, 1887.
8. Garnier, C., and Grosjean, L.: Variété rare de lipome pédiculé douloureux de la région sternale, Rev. méd. de l'est **35**: 654, 1903.
9. Beyers, C. F.: A Case of "Subpleural" Lipoma in a Child, Lancet **204**: 283, 1923.
10. Fitz, R. H.: Intrapleural Lipoma; Acute Pericarditis; Pericardial Exploration, Tr. A. Am. Phys. **20**: 57, 1905.
11. Ewing: Discussion of Fitz' Case, Ibid. **20**: 66, 1905.
12. Bertoli, P.: Un raro Caso di Lipomi multipli sottopleurici nel Mediastino anteriore, Gazz. d. osp. **29**: 1108, 1908.
13. Leopold, R. S.: A Case of Massive Lipoma of the Mediastinum, Arch. Int. Med. **26**: 274, 1920.
14. Lemon, W. S.: Lipoma of the Mediastinum, M. Clin. North America **8**: 1247, 1925.
15. Morriston Davies, H.: Surgery of the Lungs and Pleura, 1930, Oxford University Press, p. 316.
16. Andrus, W. DeW.: Report of Chest Tumor Registry, J. THORACIC SURG. **4**: 236, 1934.

INTRATHORACIC CYSTIC LYMPHANGIOMA*

G. F. Skinner, M.D., F.R.C.S. (Edin.), and M. E. Hobbs, M.D. (Tor.)
Saint John, N. B., Canada

THE mediastinum harbors several types of benign tumors which are amenable to surgery. Of these the more important are the ganglioneuromas,[1] lipomas,[2] chondromas,[3] teratomas[4] and cysts. The majority of the cysts are dermoids,[5] although others have been reported from this region, such as thymic cysts, bronchogenic cysts, and cystic lymphangiomas.

The first congenital cyst of the mediastinum recorded in medical literature was reported by Gordon[6] in 1827. This was considered to be an aneurysm until it ruptured and discharged contents typical of a dermoid. During the following fifty years very few were recorded. The first case to be operated on was a dermoid which Pohn[7] drained through the neck in 1871. In 1889 Godlee[8] in England incised and drained one. Others were reported before the end of the last century. There was very little interest, however, in this condition until recent rapid development in thoracic surgery made it possible to remove completely even very large cysts by wide transpleural exposures. During the last few years so many cases have appeared in the literature that one is scarcely justified in reporting a single case, except for the fact that the majority of recorded mediastinal cysts have been dermoids or teratomas.

In 1933, after a complete review of the literature, Hedblom[5] collected 191 mediastinal dermoids. About a quarter of these were cured by complete extirpation. Aside from dermoids few cases of congenital mediastinal cysts have been recorded. Bronchogenic cysts have recently been reported by Lilienthal,[9] Eloesser,[10] and Mixter and Clifford.[11] The latter have also found cysts of gastrogenic origin. The possibility of thymic cysts and cystic lymphangiomas has been mentioned by Ewing,[12] Nasse,[13] and Lilienthal,[14] but few cases of mediastinal cystic lymphangioma have been discussed in the recent literature. In fact, in a rather thorough survey of the literature for the past ten years, the only cases found were the ones reviewed and recorded by O. Michaelis[15] in 1934, who mentioned seven cases in his discussion and reported one of his own.

*From the Saint John Tuberculosis Hospital.
Received for publication April 1, 1936.

CASE REPORT

G. Y., white male, aged seven years, English, was admitted to hospital on June 25, 1934. The parents considered that the boy was always delicate. Past illnesses were of no consequence. Symptoms referable to the chest commenced at the age of four, consisting of shortness of breath, undue fatigue, poor appetite, and slowness in eating his meals. In May, 1933, the boy developed a very troublesome nonproductive cough, often followed by vomiting. Whooping cough was diagnosed, and the child was put to bed for three weeks. The cough persisted, and in May, 1934, the symptoms were so intense that it became necessary for him to assume the orthopneic position. The boy complained of marked tightness in his chest, always

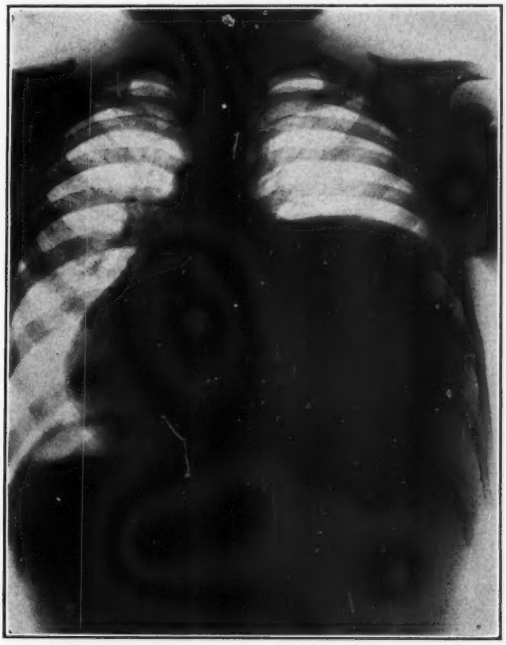


Fig. 1.—Roentgenogram of the chest after removal of 690 c.c. of fluid, showing the smooth upper border of the mass in the right side of the chest. The diaphragm is entirely obscured on this side.

increased by lying down. The diagnosis previous to admission was right-sided pleurisy with effusion.

On admission the temperature was 98°, respiration 22 to 28, and pulse 92 to 100. He was 13 pounds underweight, obviously anemic, but mentally alert. The superficial veins of the entire body were much more prominent than normal, this feature being most marked over the head, arms, and upper chest. The right side of the chest was larger than the left, the ribs being elevated, and respiratory movement considerably diminished. The maximum intensity of the apex beat was in the sixth left interspace in the anterior axillary line. Over the right side of the chest tactile and vocal fremitus were increased, and the percussion note was flat from the second rib to the base anteriorly, and from the seventh vertebral spine to base posteriorly. Breath sounds were absent over this area. The left lung was normal.

There was no abnormality in the heart sounds. The liver edge was palpable three fingerbreadths below the right costal margin on deep inspiration and was smooth and not tender. The remainder of the physical examination showed no abnormality.

Laboratory Data.—Urine, blood Wassermann and Kahn tests were normal. Blood: red cells were 4,600,000; hemoglobin 69 per cent; and white cells 8,900; differential count, neutrophiles 72 per cent, eosinophiles 2 per cent, lymphocytes 23 per cent, monocytes 2 per cent and basophiles 1 per cent.

Fluid aspirated from the right chest was at times clear and at other times dark brown or sanguineous, containing many red cells. Repeated cultures were sterile,

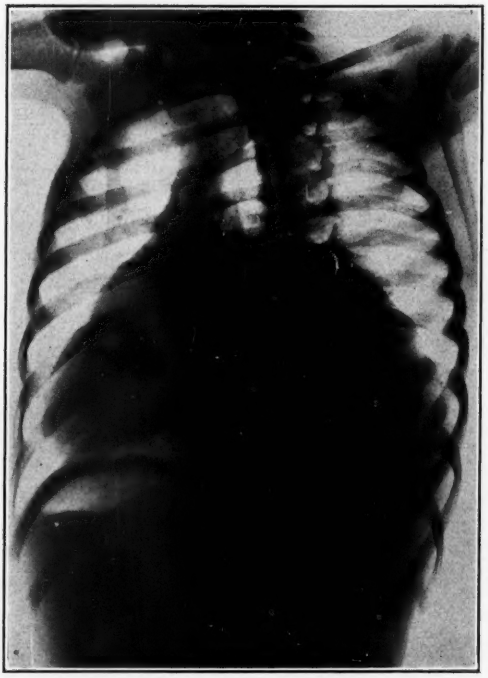


Fig. 2.—Roentgenogram of the chest showing a lateral view of Fig. 1. It is seen that the mass is mainly anterior in the right side of the chest.

and guinea pig inoculation was negative for tuberculosis. Examination revealed no hair, echinococcus hooklets or cholesterin crystals.

Roentgen Ray Examination.—Roentgen ray of the chest on June 26, 1934, (Fig. 1) showed marked clouding of the right side of the chest with fluid extending almost to the third rib. The heart and mediastinal structures were pushed to the left. The lateral view (Fig. 2) revealed the fact that the mass was mainly anterior in the chest and had smooth rounded margins. This was thought to be an unusual appearance for pleural effusion, and an intrathoracic cyst was suspected. Fluoroscopic examination showed that the mass moved downward and to the left on inspiration.

On September 18, 355 c.c. of sanguineous fluid were withdrawn from the mass, and 100 c.c. of air introduced with the needle in the same position, that is, in the cystic mass. Following this the boy was turned on his face and 300 c.c. of

air introduced into the right pleural cavity at a pressure of +10 mm. of water. With air in both the cyst and the free pleural space it was now evident that there was a fluid level in the cyst which did not communicate with the pleural cavity. This was verified by inverting the patient (Fig. 3). The tumor left the diaphragm and was seen to move freely. A diagnosis of probable dermoid cyst with mediastinal attachment was made. At this stage the only other possibility considered was a bronchogenic congenital cyst. The esophagus was outlined with barium and seen to be somewhat deviated to the left. Thoracotomy was decided upon, and pneumothorax was maintained as a preliminary measure.

First Operation.—On November 6, 1934, avertin was given, and an exploratory major thoracotomy was performed with nitrous oxide and oxygen anesthesia with positive pressure.

Fig. 3.—Roentgenogram of the chest after induction of pneumothorax on the right, taken with the patient inverted. The arrows A mark the outer edge of the collapsed lung. The arrows A_1, mark the edge of the large cyst wall. The arrows A_3, mark the edge of the inner solid portion of the cystic tumor. There is no fluid in the pleural space. The small amount of fluid in the cyst is not well shown in this picture. The mass has been separated from the diaphragm by this posture.

An incision 6 inches long followed the course of the seventh rib. Small segments of the sixth and seventh ribs were removed from their posterior ends. The incision of the parietal pleura was made between the seventh and the eighth ribs and was nearly as long as the skin incision. No respiratory embarrassment followed. There was a small amount of clear pleural fluid. The upper lobes of the lung were pinkish and partially aerated while the lower lobe was dark red and completely collapsed into the paravertebral region. Most of the exposure was filled with the large multiloculated cyst. Except for minor adhesions the cyst had no connection with the lung.

The cyst was free in front, laterally and posteriorly. On these surfaces it was covered with parietal pleura. The diaphragmatic surface was free except in its medial third. The medial surface of the cystic tumor was firmly adherent in its whole length, especially to the pericardium and the vena cava. It not only completely covered the right lateral surface of the pericardium, but also extended partially across the front of it. The mass was so adherent that it was decided not to try to remove it in one stage. Some of the larger cysts were aspirated so as to relax the tumor sufficiently to draw it into the anterior end of the incision. The aspirated fluid from some of the locules was pale, whereas from others it was dark brown. All the fluid was thin and watery.

The anterior portion of the multiloculated cyst thus relaxed was drawn into the anterior end of the incision and marsupialized. A drainage tube was inserted

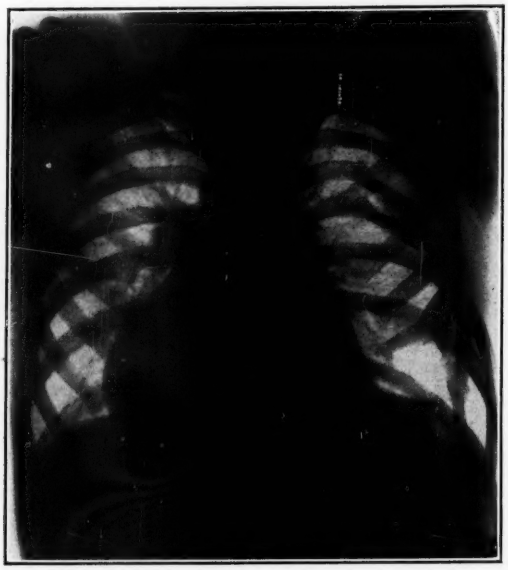


Fig. 4.—Roentgenogram of the chest six months after removal of the cystic tumor. The lung has reexpanded, and adhesions are evident in the pericardiophrenic angle.

into the lowest part of the pleural cavity through a stab wound. The posterior three-quarters of the incision was closed, drawing the ribs together with chromic catgut sutures. The anterior quarter of the incision was occupied by the exteriorized portion of the cystic mass.

Nothing of consequence occurred during the postoperative course following this first operation. One of the drained cysts about 2 inches in diameter remained partially exteriorized.

Second Operation.—On November 29, 1934, the child's condition was considered good enough to warrant a second operation. The intention was to reduce the size of the tumor by opening several of the cysts, working from within the mass without opening the pleural cavity, and then possibly to remove the contracted remaining cyst wall at a third operation. The skin incision was made to encircle the exteriorized area and to extend forward and upward to the border of the

sternum. About one and one-half inches of rib and one and one-half inches of cartilage were removed extrapleurally from both sixth and seventh ribs, that is, from above and below the exposed area of cyst. The tumor mass was now further opened through the exposed or exteriorized part. Another cyst was thus emptied without entering the pleural cavity. This procedure was repeated and a further cavity was entered. An aspirating needle was used each time as a guide, and, if fluid was withdrawn, the needle tract was followed with the scalpel. In attempting to open a third cyst the pleural cavity was opened.

Since we had now entered the free pleural space, we determined to make an attempt at radical extirpation. To increase the exposure the incision was carried

Fig. 5.—Photograph of the tumor after removal of the fluid contents of practically all the loculated cysts.

upward along the sternum, and small portions of the fourth and fifth ribs and cartilages were removed. Thus the final skin incision was the anterior third of the previous posterior incision used at the first operation, carried forward to the sternum and upward to the cartilage of the third rib. When the pleura was incised the full length of this incision, the exposure was excellent, very little retraction being necessary. After fresh adhesions to the lung, pleura and diaphragm were freed, the cyst was found to be just as previously described. No line of cleavage could be found on the medial surface. Hence we continued opening more cysts, with one hand behind the mass as a guide for the aspirating needle, and following the exploring needle with the scalpel. Clear fluid was found, pale from some and dark brown from others. With this procedure the tumor became relaxed and free, except for the adherent medial surface.

Eventually it was decided that another fairly large cyst remained unopened against the pericardium in the middle of the mass. This was opened, and through this a plane of cleavage was found. The medial surface was then freed, and a small area was separated from the medial part of the diaphragm by sharp dissection. This left only a pedicle-like extension upward to the region of the thymus, and the attachment to the vena cava. The former was clamped, divided and ligated and the latter freed by sharp dissection. A few small cysts remained in the angle between the pericardium and the diaphragm. These were destroyed after removing the large mass. The incision was closed completely, after the insertion of a stab wound drain in the lowest part of the pleural cavity.

The immediate postoperative condition was quite satisfactory. Drainage from the pleural cavity was small in amount and serosanguineous in character. The intrapleural pressure was maintained between −25 to −10 mm. of water. The pleural drainage was discontinued on the fifth postoperative day. On the ninth day the temperature rose to 103° (rectal), and a few days later aspiration revealed a localized staphylococcic empyema on the operated side. On December 21, creamy pus was obtained from a small pocket of empyema, and open drainage was instituted. The temperature fell quickly to normal. On December 29, 1934, an indirect transfusion of 300 c.c. of blood was given for secondary anemia. Improvement became rapid. Pleural drainage stopped on February 3, 1935, and on February 11, 1935, the boy was allowed up. By June, 1935, he had gained eight and one-half pounds and was symptom free. At this time roentgen ray (Fig. 4) showed the lung reexpanded. Up to the present date, July, 1936, there has been no recurrence of either cyst or empyema, and no herniation of the wound.

Gross Pathology.—In situ the mass was a multiloculated extrapleural cyst almost filling the lower half of the right side of the chest. On its medial surface it was firmly adherent to the structures of the anterior mediastinum, the pericardium and vena cava. The medial surface not only was firmly adherent to the pericardium but extended partially across the anterior surface of the pericardium. Its anterior, lateral, and posterior surfaces were covered by parietal pleura as was the lateral two-thirds of its diaphragmatic surface. The tumor was ovoid in shape with its small end upward; about five inches in its greatest diameter and at least six inches long. The individual cysts varied in size from a pinhead to two inches in diameter. The wall of some cysts was thin, but of others thick and trabeculated. The thicker walls between the large cysts contained numerous minute ones. The content of most of the cysts was thin, watery, and pale but in some cases dark brown and watery, while in a few small ones, the content was gelatinous yet translucent. The only part of the cyst with blood vessels that had to be clamped in removing it was the upper end, extending toward the thymus. This we concluded was the pedicle. No hair or sebaceous material was found in the cysts.

Microscopic Sections.—Twelve different sections showed only hyaline fibrous tissue with scattered clusters of fat cells, foci of lymphocytes or small round cells, blood vessels, scattered areas of smooth muscular fibers, and large and small cystic areas lined with a single layer of flat endothelium. Areas from which cholesterol crystals had been dissolved were also seen.

Diagnosis.—Cystic lymphangioma (hygroma cysticum).

COMMENT

In the preoperative history the chief point of interest was the conclusive diagnosis of a cystic tumor. This was made possible by roentgen ray following the injection of air into both the tumor and the free pleural space. Roentgen rays taken with the patient inverted (Fig. 3) were particularly instructive.

In the operative removal of the tumor the most striking facts were the poorly defined limits of the tumor and the lack of clear-cut planes of cleavage. The words of Michaelis[15] express our findings very accurately: "While it is usually possible to succeed in the complete extirpation of dermoids and ciliated cysts because of their well-defined borders, it is almost impossible to remove cystic lymphangiomas intact on account of their ill-defined borders. Numerous small cysts are formed in the depths and these make a shelling out of the tumor as a whole, very difficult." This very aptly describes the clusters of minute cysts remaining in the angle between the diaphragm and the pericardium after we had removed the large mass.

The real interest of the case, however, is the pathologic diagnosis. With the vast majority of mediastinal cysts reported as dermoids or teratomas, we immediately wondered if this was not just another such tumor with a one-sided development. Yet there is nothing in the structure of this tumor that is not compatible with a simple tumor, a lymphangioma. It would seem against the general principles of pathology to use a complex explanation for a tumor of simple structure. Fat and smooth muscle are not at all unusual in lymphangiomas (Ewing). And areas of cholesterol crystals confined to the wall rather than in the lumen are found in such cystic tumors as the result of degeneration of the islands of fat (Wood[16]).

Although such tumors are probably not teratomatous, they are very likely of congenital origin. They are due to a late isolation of mature differentiated cells rather than to the early segregation of a premature type of cell, as in teratomas.

It has been suggested that intrathoracic lymphangiomatous cysts are cystic hygromas which have grown down from the neck (Eigler[17]). In a case such as the one reported here it is quite conceivable that a portion of the anlage for vessel formation could be drawn down from the region of the gill cleft by the pericardium in its descent. The intimate relation of the cystic tumor to the pericardium certainly suggests an early association.

Another possibility would be a thymic origin, and in this case we cannot rule out such a source, for although the pedicle was from the region of the thymus, the actual relation of it to the thymus was not seen.

A local origin of such cysts due to an inflammatory process or to stasis has been discussed by Michaelis,[15] but he dismisses the idea as most improbable.

Histologically the case here reported, like the one recorded by Michaelis, is distinctly a cystic lymphangioma. It shows no evidence of being a mixed tumor or teratoma. The only question seems to be its relation to the thymus. As a lymphangioma it could arise from

the thymus or directly from a branchial cleft anlage. But since the thymus is derived from a branchial cleft, the ultimate origin would be the same in either case. From this high origin in the neck such a tumor would be drawn into the thorax by the descent of the heart.

Much confusion exists as to the classification and nature of intrathoracic cysts. This is probably due to the great preponderance of dermoids and teratomas, which fact has led to a tendency to try to classify all mediastinal cysts as such, by presupposing a one-sided development in doubtful cases which show structures derived from only one primary tissue type.

Most recent observers in discussing mediastinal tumors do not mention cystic lymphangiomas. Ewing and Lilienthal both include them as possibilities but discuss them mostly as types of thymic cysts. Ewing quotes a case reported by Seidel[18] in which the thymus was replaced entirely by a cystic lymphangioma.

Sauerbruch[19] in 1925 classified mediastinal cysts as follows:

1. Dermoids.
2. Cysts lined with ciliated epithelium.
3. Cystic lymphangioma.

He stated that the last named are the most uncommon. This would seem to be a fair classification, but possibly it would be better to say columnar cell epithelium rather than ciliated, to include the gastrogenic cases reported by Mixter and Clifford.[11]

A similar classification, but one using more general terms, might be helpful at present.

1. Ectodermic cysts —simple dermoids—epidermoids.
2. Entodermic cysts —bronchogenic, gastrogenic.
3. Mesodermic cysts —cystic lymphangiomas.
4. Mixed cysts —complex dermoids, teratomas.
5. False cysts —parasites, degenerate solid tumors, calcified hematomas (Wright[20]), etc.

SUMMARY

1. A large mediastinal cystic lymphangioma is reported in detail.

2. Roentgen ray diagnosis of a cystic tumor was made after injection of air into both the tumor and the pleural cavity.

3. Complete extirpation of the tumor was accomplished through an anterolateral incision.

4. The patient is alive and well nearly a year and a half later, and there is no sign of recurrence of the tumor.

5. The pathologic diagnosis and the classification are discussed briefly.

REFERENCES

1. Sophian, L.: Ann. Surg. **101**: 827, 1935.
2. Heuer, G.: Ann. Surg. **98**: 801, 1933.
3. Heuer, G.: Arch. Surg. **18**: 273, 1929.
4. Harrington: J. THORACIC SURG. **3**: 50, 1933.
5. Hedblom, C. A.: J. THORACIC SURG. **3**: 23, 1933.
6. Gordon, J. A.: Med. Chir. Transactions, London **13**: 12, 1827.
7. Pohn: Inaug. Dissert. Berlin, 1871.
8. Godlee, R. J.: Med. Chir. Transactions, London **72**: 317, 1889.
9. Lilienthal, H.: Arch. Surg. **18**: 292, 1929.
10. Eloesser, L.: Surg. Gynec. Obst. **52**: 747, 1931.
11. Mixter and Clifford: Ann. Surg. **90**: 714, 1929.
12. Ewing, J.: Neoplastic Diseases, Philadelphia, 1928, W. B. Saunders Co.
13. Nasse: Langenbecks Arch. f. Chir. **38**: 614.
14. Lilienthal, H.: Thoracic Surgery, Philadelphia, 1925, W. B. Saunders Co.
15. Michaelis, O.: Deutsche Ztschr. f. Chir. **242**: 250, 1934.
16. Wood, F. C.: Nelson Loose Leaf Surgery, Vol. 2, p. 45.
17. Eigler: Dtsch. Z. Chir. 189, 1926.
18. Seidel: Inaug. Dissert., Leipzig, 1904.
19. Sauerbruch: Chirurgie der Brustorgani Bd. 2, Berlin, 1925.
20. Wright: Brit. J. Surg. **23**: 612, 1936.

UNUSUAL COMPLICATIONS FOLLOWING CLOSED INTRAPLEURAL PNEUMOLYSIS*

Report of Two Patients

James H. Forsee, M.D.†
Denver, Colo.

THE more important complications following closed intrapleural pneumolysis are: pleural effusion, superimposed spontaneous pneumothorax, and hemorrhage.[1, 2] In the two patients being reported, complications occurred which are infrequently reported, and I believe they are of sufficient interest to warrant recording. In both, thoracoscopy revealed adhesions considered suitable for cauterization by the electrosurgical method. There was no departure from our usual operative technic; neither patient presented unusual operative difficulties, and both patients were returned to their wards in good condition. In the first case reported, a massive contralateral tuberculous pneumonia developed. In the second case, persistent severe hiccough developed seven days postoperatively.

CASE REPORTS

Case 1.—J. H. F., aged thirty-two years, soldier, was admitted June 27, 1933. Far advanced pulmonary tuberculosis with a 4.5 cm. cavity left upper lobe. Artificial pneumothorax, left, induced July 13, 1933. Pleural adhesions prevented closure of the cavity (Fig. 1). Thoracoscopy Feb. 7, 1934, revealed three adhesions extending from a common site on the partially collapsed upper lobe to the area of the fourth rib and intercostal space extending from the mid to the posterior axillary line. These adhesions consisted of a string, a fan-shaped, and a densely fibrous band, 2 cm. wide and 1 cm. thick, the latter being attached posteriorly. These adhesions were severed by the electrosurgical method, following which a string and small thin fan-shaped adhesion were seen attached posteriorly at the fifth intercostal space. They were very taut and were severed with resultant increase in pulmonary collapse (Fig. 2).

There were no untoward developments until fourteen hours postoperatively (2 A.M.), when the patient's condition suddenly became critical.

*From the Medical and Surgical Services, Fitzsimons General Hospital. Published by permission of the Surgeon General, U. S. Army.

†Captain, Medical Corps, U. S. Army.

Received for publication, March 4, 1936.

Pulse 140, respiration 4-5 per minute, lips cyanotic, and patient was deeply comatosed. Air was removed because of positive intrapleural

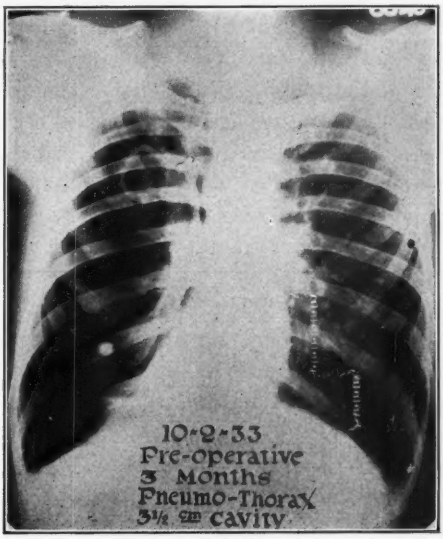


Fig. 1.

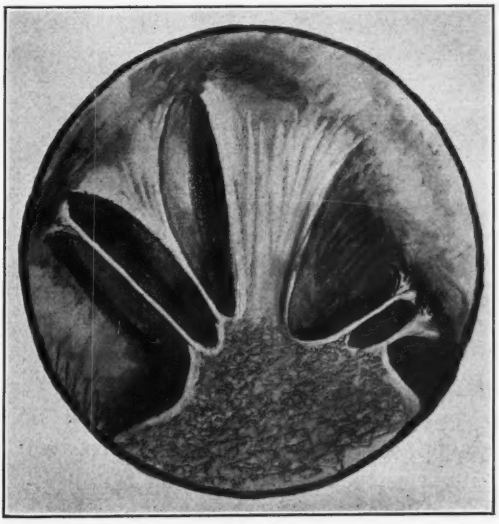


Fig. 2.

pressure, left. Treatment for shock was instituted immediately, including glucose intravenously. At 5 A.M. he was rational, blood pressure

90/60, respiration 20. At 7:30 P.M., the retention needle was removed, and no change occurred until midnight when he again suddenly lost consciousness and presented the clinical picture as seen on the previous night except more severe. Similar therapy failed to bring about the desired response and great difficulty was experienced in maintaining respiration. The placing of the patient on the nonoperative side and intermittent traction on his tongue were the only means by which he could be kept breathing. After about five hours he regained consciousness; oxygen therapy was required for forty-eight hours, and the retention needle was not permanently removed for eight days.

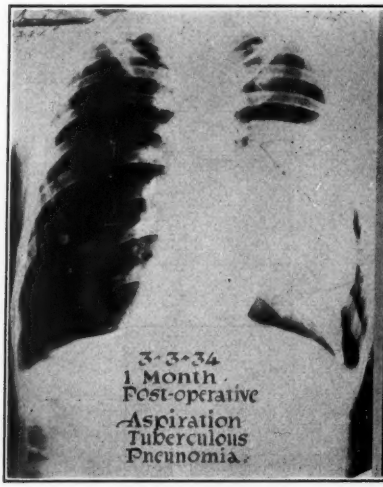


Fig. 3.

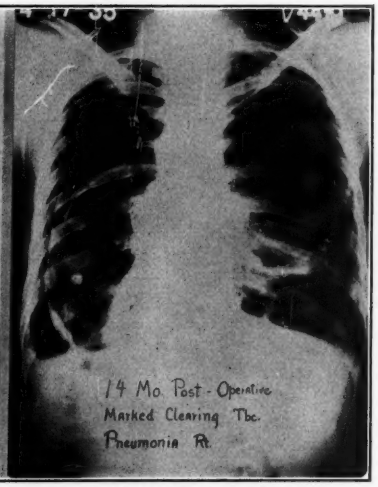


Fig. 4.

Elevated temperature (100° F.) and rapid pulse continued with a marked increase in the amount of sputum, and within three weeks there was a massive consolidation of the lower three-fourths of the right lung. Artificial pneumothorax was induced on this side Feb. 28, 1934. This therapy was not instituted in an attempt to compress the consolidated tuberculous lung but rather to establish a pneumothorax space before pleural symphysis could occur so that the desired lung compression could be accomplished as the pneumonic process resolved (Fig. 3).

An excellent pneumothorax on the left resulted following the severing of adhesions, and the cavity closed within two months. The complication of an ulcerative tuberculous laryngitis developed. A small hydropneumothorax, left, appeared five months after operation and disappeared after a few aspirations. Fluid on the right has appeared in

small amounts from time to time, beginning eight months after operation. Phrenic exeresis, right, was performed May 5, 1934, as an adjunct to the pneumothorax therapy.[3]

The tuberculous pneumonia gradually cleared without excavation of lung parenchyma (Fig. 4). The patient left the hospital in October, 1935, with recommendations to continue the bilateral pneumothorax. The tuberculous laryngitis was greatly improved, ulceration healed, and there was some hoarseness of voice. No cavitation was present, the sputum had been negative for eighteen months, and his general condition was much improved.

DISCUSSION

The mechanism of the production of the massive pneumonic consolidation in the contralateral lung warrants discussion. The sudden loss of consciousness with a markedly decreased respiratory rate is not the usual course of events in a superimposed spontaneous pneumothorax, as there is generally a period of conscious dyspnea. The suddenness with which the condition became critical suggests a severe respiratory embarrassment, mediastinal displacement and disturbance of cardiac function producing severe shock. The mechanism of the contralateral pneumonia is, I believe, the result of a bronchogenic aspiration from the operated side.[4] During the second postoperative night, secretions could be seen filling the upper portion of the trachea; and with the patient lying on his so-called good side, bronchogenic dissemination of the secretions into the better lung appears to be a logical explanation. There were scattered tuberculous lesions in the so-called good lung which appeared fibrous and were unlikely the source from which a massive pneumonic involvement could be produced.

The rapidity with which the condition became critical may be explained on the basis of a profound shock resulting from the production of a patent bronchopulmonary pleural fistula with marked disturbance in the intrapleural pressure and displacement of the mediastinal structures to the contralateral lung.

This case well illustrates the benefits of artificial pneumothorax in the treatment of tuberculous pneumonia. The condition developed under close observation, and the pneumothorax was instituted at a selected time. The purpose for which the pneumothorax was established has been discussed. This idea of effecting the desired lung compression as the pneumonic process resolved was carried out, and, considering the extent of the involvement, the result was very satisfactory. The phrenic exeresis, right, was and will be of decided benefit when the lung is allowed to reexpand.

CASE 2.—J. E. C., aged thirty-seven years, soldier, was admitted March 17, 1934, with far advanced bilateral pulmonary tuberculosis.

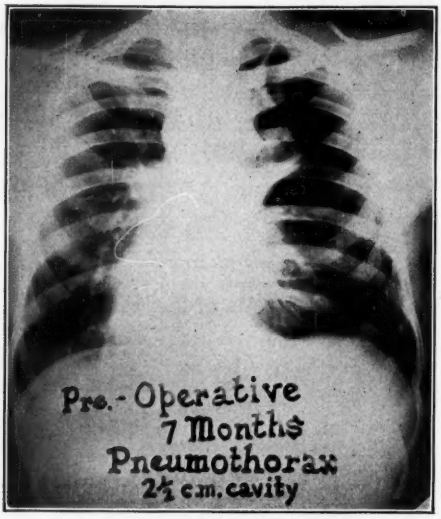


Fig. 5.

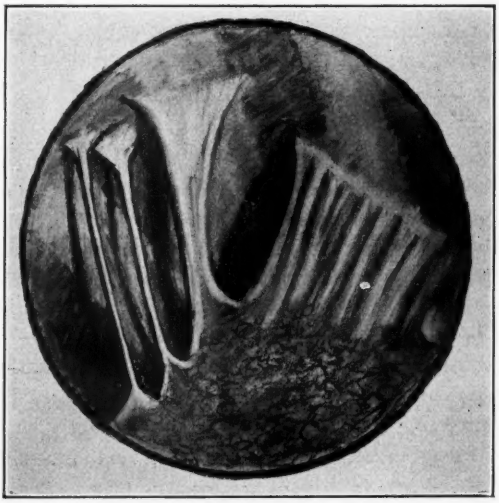


Fig. 6.

Artificial pneumothorax, right, had been induced at Mare Island, California, seven months prior to admission. A 2.5 cm. cavity remained patent because of pleuropulmonary adhesions (Fig. 5). Thoracoscopy

June 27, 1934, revealed two string and one small cord adhesions extending from the partially collapsed lung to the second intercostal space in the mid and posterior axillary line. More posterior was a 4 cm. band adhesion blending indistinctly with adherent lung tissue. The string and cord adhesions were severed, and the band adhesion was coagulated along its parietal pleural attachment with the coagulation extending deep into the adhesion (Fig. 6).

The postoperative course was uneventful until the fourth day when an acute pleuritis developed and 350 c.c. of clear fluid was aspirated. Two days later, July 4, severe hiccough began. Exhaustive medication was without effect except for temporary relief following inhalations of

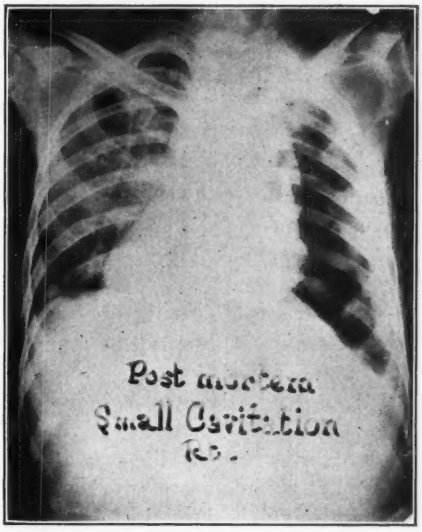


Fig. 7.

carbon dioxide and oxygen.[5] Fluoroscopic study showed bilateral spasmodic contractions of the diaphragm, more pronounced on the right. The patient's condition became critical. Phrenic exeresis, right, and alcohol injection of the main trunk of the left phrenic nerve were performed July 7. The hiccough continued but was less severe, and gradually there developed periods of a few hours when this condition was absent. It was not, however, until nine days after the onset, July 13, that it had completely disappeared. Reexamination fluoroscopically twenty-four hours after operation showed bilateral spasmodic contraction of the diaphragm but to a lessened degree. During this interval the contralateral involvement increased, the pleural effusion became purulent, and a *Staphylococcus aureus hemolyticus* was cultured from the exudate.

Frequent aspiration, irrigation, and instillation of aqueous gentian violet solution, 1 to 5 per cent, were instituted. The amount of exudate gradually decreased, and repeated cultures of the fluid over a period of ten months were negative. Attempted reexpansion of the collapsed lung was unsuccessful and pleurocutaneous fistulas developed. Retrogression continued, temperature 99-101° F., pulse 90 to 120, occasional nausea and vomiting, and myocardial damage became evident. Thoracoplasty was indicated to decrease the size of the empyema cavity and to compress the diseased lung, but the patient remained too ill for operation. The liver became markedly enlarged and tender. The patient died Dec. 23, 1935 (Fig. 7).

Autopsy revealed generalized amyloidosis of the liver, spleen and kidneys. Small cavitation was present in the upper lobe of the collapsed lung. The band adhesion coagulated at operation was elongated and very dense. No tuberculous enteritis was found.

DISCUSSION

Hiccough following operation upon the chest is much less frequent than in upper abdominal surgery. In this case, its occurrence six days after closed intrapleural pneumolysis in which an acute pleuritis developed is unusual. What part the latter condition had in the production of the hiccough is unknown. Frequently, patients under pneumothorax therapy develop an acute pleuritis, but hiccough is a rare complication. It has not previously occurred in my cases of intrapleural pneumolysis.

The treatment of this complication was, as is frequently the case, relatively unsatisfactory. Bilateral phrenic nerve paralysis did not eradicate the spasmodic contractions of the diaphragm. Alcohol injection of the main trunk of the left phrenic nerve was, however, probably ineffective in completely paralyzing the hemidiaphragm because of accessory phrenic nerve intervention. The patient was very apprehensive regarding surgery; and, following his removal to the operating room for phrenic nerve surgery, he was completely free of hiccough for one hour before and during operation. It returned, however, thirty minutes after return from the operating room. The severity of this complication was an important factor in lowering this patient's resistance. When it became evident that the lung could not be reexpanded, extrapleural thoracoplasty was indicated. This procedure was not carried out early because it was believed treatment of the empyema would make him a better surgical risk. His continued retrogression and final development of amyloidosis, however, precluded operation.

SUMMARY

Two unusual complications following closed intrapleural pneumolysis have been presented. In one patient a severe contralateral tuberculous

pneumonia developed. Its mechanism was that of a bronchogenic aspiration following a superimposed spontaneous pneumothorax and severe shock. The early establishment of an artificial pneumothorax of this side and the accomplishment of the desired lung collapse as the pneumonia process cleared gave excellent results. In the second patient, persistent severe hiccough developed six days postoperatively and continued for nine days. Exhaustive medication including CO_2 and oxygen inhalations was ineffective. Unilateral phrenic exeresis and alcohol injection of the main trunk of the phrenic nerve on the opposite side did not eliminate the spasmodic contractions of the diaphragm. An empyema of a mixed infection type ensued which did not respond to treatment sufficiently to permit major surgery. Amyloidosis developed, and the patient died.

REFERENCES

1. Forsee, J. H.: Closed Intrapleural Pneumolysis, Ann. Int. Med. 8: 1309, 1935.
2. Moore, J. A.: Intrapleural Pneumolysis, a Critical Review, J. THORACIC SURG. 3: 276, 1934.
3. Pollock, W. C., and Forsee, J. H.: Phrenic Exeresis in Conjunction with Artificial Pneumothorax Therapy, J. THORACIC SURG. 4: 509, 1935.
4. McCordock, H. A., and Ballon, H.: The Mechanism of the Development of Tuberculous Pneumonia Following Thoracoplasty, J. THORACIC SURG. 2: 24, 1932.
5. Sheldon, R. F.: Control of Hiccup by Inhalation of Carbon Dioxide, J. A. M. A. 89: 1118, 1927.

In Memoriam

JOHN SMYTH
1869—1935

John Smyth was born November 16, 1869, in Tensas Parish, La. He descended from a long line of distinguished Scotch-Irish ancestors. His uncle, Dr. Andrew Woods Smyth, attained great prominence as house surgeon of Charity Hospital, New Orleans, and is recalled particularly because of having performed the first successful ligation of the innominate artery in 1864. Another uncle, Dr. William Woods Smyth, was a distinguished ophthalmologist and personal physician to King Edward VII of England.

Doctor Smyth's early education was directed toward becoming a civil engineer, graduating from that department of Tulane University, in 1886, which profession he then followed for ten years. At the age of twenty-seven years, in 1896, he entered the Medical School of Tulane University. His previous education, practice, and experience in engineering served as a most admirable preparation for the scientific study of medicine and surgery. He received his degree in medicine in 1900. For the succeeding nine years he was affiliated with Dr. Matas, during which time he applied his knowledge of physics and mechanics in the development and utilization of the roentgen ray for diagnostic and therapeutic purposes and with which he obtained results that were amazing when his rudimentary equipment is considered. His many contributions during this period demonstrated his great technical skill and discriminating knowledge of the potentialities and limitations of this modality, far in advance of the experience of his time.

In 1902 he was appointed instructor-in-chief of the Laboratory of Minor Surgery which was, at that time, established by Dr. Matas, and rose rapidly by successive steps to become assistant professor of Clinical Surgery. In 1917, he entered the World War, serving on the surgical staff of Base Hospital No. 24, rising to the grade of major and being discharged with the rank of lieutenant colonel. He was honored by receiving the United States Military Cross. After the war he returned to Tulane University and was appointed professor of Clinical Surgery.

While interested primarily in roentgenographic technic and roentgen therapy, Dr. Smyth demonstrated his mechanical genius and inventiveness also in experimental research in physiologic and pathologic effects of positive and negative atmospheric pressure on the lung and respiratory function, in states of pulmonary collapse induced by surgical

pneumothorax. These studies, which were undertaken in 1901 under the direction of Dr. Matas, culminated in the perfection of an apparatus which furnished basic data for accurate and rational application of intralaryngeal insufflation.

In 1900, also at the suggestion of Dr. Matas, he conducted a series of clinical studies, on volunteer medical students, to determine the effects of Momburg's method of aortic compression by circular elastic abdominal constriction which demonstrated the dangers of this procedure.

In addition to his earlier writings on roentgen technic and therapy and the mechanics of artificial respiration in thoracic surgery, he wrote extensively on bone transplantation for closure of cranial vault defects, fractures, arthroplasty, hernia, gastric and duodenal ulcers, gastroenterostomy, oral anesthesia, interpretation of abdominal pain, and endoaneurysmorrhaphy.

During the last thirty-three years of his life, the bulk of his practice was centered at the Hotel Dieu. His skill is remembered gratefully by countless patients, and the memory of his charity and devotion to them will probably be his most enduring monument.

Though not gregarious in his tendencies and always retiring, unpretentious and reserved, he became early a member of the local, state, and national societies. He was a Fellow of the American Association for Thoracic Surgery from 1920 to his death (1935); of the Southern Surgical Association, American College of Surgeons, Association of Military Surgeons, and correspondent of the Edinburgh Research Society.

In 1911 he married Miss Jeane Sully and is survived by his widow and two sisters. His fatal illness was caused by a hypernephroma, which caused complete portal obstruction.

After an intimate friendship of thirty-seven years, the writer mourns the loss of Dr. Smyth as a personal calamity. For he was true as steel, the incarnation of honesty, the soul of honor and unswerving loyalty. Very discriminate in his friendships, his affection and fidelity to those to whom he was attached were indeed a prized privilege and a great compliment.

As a surgeon he was conscientious and cautious to an extreme degree, fastidiously meticulous in every detail for the patient's safety, anxious to do no harm if he could do no good. His sense of responsibility so worried him before and after any serious operation that it imposed great hardship upon his energy and sensibilities by his sleepless vigilance. His solicitude and care for his patients, poor or rich, won their confidence, and rewarded him by their grateful affection and friendship.

In his professional relations with his colleagues, Dr. Smyth was always courteous and punctilious in observing the Golden Rule, treating others as he wished them to treat him.

He met death like a true doctor. Conscious almost to the last, he bore his trials with a calm and stoic fortitude, an equanimity of spirit characteristic of his courage, and becoming a physician who had met Death and stayed his hand too often to be fearful of his presence when he came, not as a foe, but as a friend to ease him of the pangs of a disease which he knew was beyond all power of human help to conquer. He passed away painlessly and in peaceful slumber, attended by the loving hands of those dearest and closest to him.

R. Matas.

William Francis Honan
1866—1935

William Francis Honan was born in Maysville, Kentucky, November 19, 1866. He died suddenly while playing golf October 6, 1935, at Huntington, L. I.

His early education was received in private schools and at the Maysville (Kentucky) Institute conducted by the famous German, Professor Caphas. He obtained his medical degree from the New York Homeopathic Medical College and Flower Hospital in 1889; he interned at Cumberland Street Hospital, Brooklyn. Two years later he began the practice of medicine and became demonstrator of anatomy at his alma mater. Dr. Honan's subsequent activities represented a wide and successful field of endeavor in surgery. He was attending surgeon at the Hahnemann Hospital—later Fifth Avenue Hospital—from 1900 to 1927, and chief surgeon of the Carson C. Peck Memorial Hospital of Brooklyn from 1918 to 1924, after which date he continued as consulting surgeon in both institutions. He was clinical professor of surgery at Metropolitan Hospital from 1896 to 1921 and then was director of surgery for several years. In 1929 he was made director of thoracic surgery at Metropolitan Hospital and at Sea View Hospital, Staten Island, positions which he retained until 1934 when he was automatically retired because of having reached the prescribed age limit.

During the war Dr. Honan organized Base Hospital No. 48 in this country, serving as its chief surgeon and commanding officer at Mars-sur-Allier, France. Commissioned major, originally, he was soon promoted to lieutenant colonel. Subsequently he was promoted to the rank of colonel in the Medical Reserve Corps, General Hospital No. 81. It was his war experience with chest cases that first aroused Dr. Honan's interest in the possibilities of thoracic surgery. After the war Dr. Honan especially devoted himself to private research and clinical studies in this field. It was in line with this interest that he devoted himself to the direction of thoracic surgery at the Metropolitan and Sea View

Hospitals, 1929 to 1934. "This work," writes one who knew Dr. Honan intimately, "was his great joy—a joy in spite of the arduous labor and inconvenience involved."

During the late summer and fall of 1924 Dr. Honan visited abroad the clinics of Dr. Bull of Norway and others in the Scandinavian countries, relative to chest surgery. In 1929 he was asked to make a survey of hospitals of the City of New York as compared with other tuberculosis hospitals in various parts of the country with the idea of submitting plans for a reorganization of the New York City Department of Hospitals for the care of tuberculosis patients. His recommendations subsequently were accepted and are incorporated in the present-day care of those cases in New York City. In 1931 Dr. Honan served as chairman of the Committee on Hospitals for the New York Academy of Medicine.

In 1914 Dr. Honan was elected a Fellow of the American College of Surgeons and in 1921 he was elected a Fellow of the Academy of Medicine. He was a member of the American Institute of Homeopathy, the New York Gastro-Enterological Society, the Medical Society of the County of New York, the Homeopathic Medical Society of the County of New York, the Homeopathic Medical Society of the State of New York, and the New York Society for Thoracic Surgery.

Dr. Honan loved his profession and held it in highest esteem, but in addition to his scientific achievements he was a man of many and enthusiastic interests. Charles Rann Kennedy, author of *The Servant in the House*, and a lifelong friend, who made an eloquent address before some 700 friends assembled at a Memorial Service in the Clinic Room of the Flower Hospital and New York Homeopathic Medical College, where Dr. Honan had for so many years lectured to students, said in part,

"He was adequate in every department of life. For instance, in my own profession, he not only was a man who had a great love of the theater, but he had a most intimate knowledge of its working and even of the art of acting. He was a splendid amateur actor. He would give an impression of Henry Irving which was really quite marvelous. Willy Francis gave a great deal of study to not plays alone, but literature. He was interested in other arts and sciences besides medicine . . . he was equally informed and alive in his heart to the simplest things. He would spend an hour telling you what a case was like. Not merely about the operation, but about the man's face and he would imitate his face, reproduce his dialogue, be interested in the man's work. He would have a plumber's feeling when serving the plumber; if it were a laundry woman he operated upon, he would immediately be interested in the science of washing and the tasks of the laundry woman . . . he would have an immediate feeling for the individual life of the person, the life they had to lead, thinking in terms of the living they had to live. In other words, he was a great artist."

William Francis Honan had a great capacity for making friends. He loved humanity and numbered among his intimates men and women in all walks of life.

A list of his contributions to surgical literature is long, but his choicest efforts and most unique achievements frequently he did not take the time to write up. Unfortunately he was one of those who would not commit himself to paper. Only his friends and intimate associates knew of his activities.

Among some of his pioneer activities in many fields of surgery it may be mentioned that Dr. Honan was the first to use intravenous anesthesia in this country and one of the first ever to wire an aneurysm. As Dr. John Alexander stated: "His sudden death at a time when he seemed to be at the height of his career and usefulness has saddened his friends and patients who had always found in him a wise and sympathetic counsellor whose kindliness and fine character endowed him with a special sort of warmth and fineness. The host of loyal patients whom his skill and personal devotion restored to health bear witness to the effectiveness of his surgical ability and his genuine desire to serve his fellowman."

Louis R. Davidson.

Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

Selected Abstracts

Pulmonary Mycotic Infections. R. A. Carter. Radiology **26**: 551, 1936.

Pulmonary mycotic infections usually simulate closely some manifestations of the very variable disease, tuberculosis. On the whole, the minority characteristics of that disease are simulated. One must think of mycosis with a persistent failure to find the tubercle bacillus together with peculiarities of the history. Geographic location, occupation, associated extrapulmonary lesions or the characteristics of the chest film may direct suspicion to mycosis or to a particular disease as blastomycosis, coccidiodes or actinomycosis.

The author lists the characteristics adding to the likelihood of a mycotic infection being present: (1) outstanding mediastinal or hilar adenopathy in an adult; (2) miliary lesions, vague or profuse, associated in the adult with adenopathy; (3) absence or sparsity of dry air-containing cavities in advanced lesions; (4) extraprominent pleural lesions, e.g., gross thickenings; (5) absence, in the adult, of an obviously older apical or subapical lesion; (6) growth of a consolidation by contiguous spread; (7) lesions of bone or soft tissue abscesses about the thorax; (8) extension of involvement through the thoracic wall; (9) hilar, truncal or peribronchial thickenings with minor parenchymal changes associated with mild chronic clinical symptoms and profuse expectoration; (10) bronchiectatic manifestations; (11) fine, discrete miliary or nodular manifestations associated with fine peribronchial accentuation in the absence of a cause for pneumoconiosis, (12) cloudy parenchymal densities accompanying a subacute persistent respiratory attack, more indefinite than in tuberculosis, which neither resolve nor evolve into tuberculous manifestations.

The author then lists miscellaneous circumstances adding to the possibility of mycosis being present when suspicious pulmonary roentgen signs are seen: (1) granulomatous nodes or ulcers in the skin, soft tissue abscesses, lesions of the bones or joints; (2) preceding respiratory attacks with slow, incomplete or complete recovery followed in a few weeks or months by pulmonary or extrapulmonary manifestations de-

scribed previously; (3) terminal meningeal symptoms; (4) geographic location, e.g., coccidiodes common in California and mycosis frequent in the tropics; (5) occupations are usually those in which there is exposure to dusts likely to contain spores of fungi, e.g., actinomycosis infection through contact with hay or grain.

Henry L. Cabitt

The End-Results of the Surgical Treatment of Pulmonary Tuberculosis. P. B. Matz. Am. Rev. Tuberc. **33**: 533, 1936.

The author reports 320 cases of pulmonary tuberculosis treated by various types of surgery. Of these cases, 56.9 per cent had had pneumothorax therapy, with or without success. Of the 320 cases, 301 were classified as far advanced, and 80 per cent of the 301 had bilateral lesions; 87.2 per cent of the original 320 cases had cavities; 50 per cent of the lesions were classified as productive; 23 per cent were exudative; 17 per cent were a mixture of productive and exudative; 10 per cent were pleural.

Phrenicectomy was helpful in lessening the cough, sputum, and hemoptysis; and cavities often diminished in extent or disappeared after operation. In spite of phrenicectomy, however, 165 out of 170 tuberculous patients died as a result of the progression of the tuberculous process. Thus, this type of operation does not save or prolong life but may be used for its palliative effects.

Eighty out of 320 patients had thoracoplasty. Twenty-seven patients were alive after an average time of 33.6 months after operation. Of these 27 patients 66.7 per cent were improved, whereas 33.3 per cent were listed as stationary. The improvement consisted of a decrease in fatigue, sputum and cough, and the closure of cavities. Fifty-three patients died of an extension of the tuberculous process in spite of thoracoplasty.

Seven patients out of 320 had intrapleural pneumolysis done. Follow-up showed that in 4 out of 6 of these cases the cavity had disappeared, and 3 patients were alive at the time of this study.

Figures show that 90 per cent of patients will be dead within five years after the appearance of a positive sputum, with treatment inside or outside of a sanatorium. Thus it would seem that surgical intervention is beneficial since it has reduced this 90 per cent mortality to 70 per cent in this series.

Henry L. Cabitt.

The "Gordon Test" for Hodgkin's Disease. J. D. Goldstein. Am. J. M. Sci. **191**: 775, 1936.

In 1932 Gordon reported an encephalitic syndrome of characteristic pattern displayed in rabbits injected intracerebrally with suspensions

of lymph glands removed from patients with Hodgkin's disease. Injections of gland biopsies from other various conditions did not give this reaction. The nature of the agent responsible for this reaction is not established although it may be a virus.

The author describes the technic of the test. Seven out of nine proved cases of Hodgkin's disease, wherein biopsy material was injected intracerebrally into rabbits, showed a positive Gordon test with a definite, specific encephalitic syndrome. As controls, suspensions of 20 lymph nodes were injected, including five tuberculous nodes and two nodes occurring in infectious mononucleosis. All these injections were negative. No false positives were obtained.

The author concludes that this test is of distinct value in diagnosis especially where the histologic findings are uncertain. At present, a negative test has no diagnostic significance.

Henry L. Cabitt.

Metastatic Tumors of Heart. J. K. Miller. Am. J. Surg., June, 1936.

Metastatic tumors of the heart are more common than primary tumors of this organ. In general autopsies they occur in 0.5 per cent of the cases. Any organ can be the primary site of the tumor. The most common primary tumor is bronchiogenic carcinoma.

Cardiac metastases are usually blood borne, occasionally due to retrograde invasion through the lymphatics, seldom by direct extension. Hematogenous dissemination infers the establishment of secondary colonies within the heart chambers and their contents. This is generally the rule. Small cellular emboli, however, may instead lodge in the terminal branches of the coronaries, forming intramural tumors. Such a case is reported by this author.

Henry L. Cabitt.

A Study of 57 Cases of Bronchogenic Carcinoma. Raymond S. Rosedale and Donald R. McKay. Am. J. Cancer **26**: 493, 1936.

Out of 466 cases of neoplasm autopsied in this series 7.5 per cent were primary carcinoma of the lung. In 91 per cent of the cases the lesion occurred in males. The age of the patients varied from 33 years to 73 years, the average age being 40.5 years. Seventy-five per cent of the patients were employed in occupations requiring exposure to dust or other irritating atmospheric factors. The lesion occurred in the right or left lung in about equal proportions.

In the majority of instances the pathologic process was present in the first order or larger bronchi near the hilus; usually as a single massive lesion in contrast to the multiple, bilateral peripheral nodules of metastatic tumors.

Histologically the tumors were composed of various predominant cell types. Round or spindle cells were found in all cases. These tumors metastasized widely; in all cases the regional lymph nodes were involved. It was repeatedly observed that massive, distant metastases arose from a small primary tumor. The commonest symptoms were cough, sputum, chest pain, and dyspnea. The duration of symptoms averaged ten months.

As to the value of x-ray diagnosis in this series, out of 30 autopsied cases with proved carcinoma of the lung, the diagnosis was made by the x-ray department in 14 cases; the diagnosis was suspected in 1 case and was completely missed in 15 cases. These errors were due, in the main, to secondary pleural or pulmonary pathologic conditions which obscured the tumor.

The authors believe firmly in the value of bronchoscopy from a diagnostic point of view. In 6 cases of carcinoma of the lung proved by autopsy, a positive diagnosis was obtained by bronchoscopic biopsy in 4 cases, and in 1 case the diagnosis was suspected. In the unautopsied group, later proved to be malignant, 3 cases gave a positive bronchoscopic biopsy, and in 2 cases pressure on a bronchus was demonstrated.

Henry L. Cabitt.